

COMPLICATIONS OF DYSENTERY

with

special reference to

ARTHRITIS.

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Introduction.

The subject of "Dysentery" is one which, from the earliest days of Medicine, has been of paramount importance to mankind; and reference is made to it by very early writers. Hippocrates, the Father of Medicine, originated the term Dysentery "*Δυσεντερία*" to denote a painful condition of the bowels associated with the passage of blood. Since his day there has been a great deal of literature on the subject, and much has been done to elucidate the intricate diseases included under that term. Much also has been done to fight the disease and to diminish its toll on the human race.

Despite the great improvements in sanitation which have done so much to lessen the ravages of the disease in all countries, it still is liable to break out during great campaigns. Thus in the Franco-German War of 1870 there were 38,652 admissions with Dysentery, with 2380 deaths; and the troops on their return spread the infection in many districts, in some of which it is still present. During the South African War, 1899-1902, there were 38,108 cases with 1342 deaths. A significant fact at that time was an outbreak of Dysentery in 1901 in Aldershot of 170 cases with 38 deaths, the disease being no doubt imported by soldiers from South Africa. In this

present, the greatest of all wars, there have been many cases of Dysentery among the armies of the various Belligerents engaged on the different fronts. Now the War is over and many men are returning to their own districts, to say nothing of the many who have been invalided on account of Dysentery and its various complications and sequelae. The present time therefore is one in which this subject assumes special importance, and no apology is required for taking this subject as a Thesis with special reference to Dysenteric Arthritis. Having been on the Staff of the General Hospital at Salonica, which specialized in Dysenteric cases, excellent opportunity was afforded me for observing the clinical aspect of Dysentery and its complications during the Winter 1917-18 and Summer and Autumn of 1918. I propose in this Thesis to give a short resumé of the subject with observations on certain cases of interest which came under my notice there.

History of the Differentiation of the Dysenteries.

The conception of Dysentery, according to ancient writers, was a disease in which there was an ulcerated condition of the bowel associated with the passage of blood and mucus in the stools and accompanied by symptoms of tormina and tenesmus. The contagious nature of the disease, which was early noticed, was accounted for by miasmata. In the middle of the eighteenth century Sir John Pringle in his work "Observations on the Diseases of the Army" in Flanders, speaking of the contagious nature of the disease, makes the following observation :-

"Having since perused the curious dissertation
"by Linnaeus, in favour of Kercher's suggestion of
"contagion by animalculae, it seems reasonable to
"suspend all hypothesis till that matter is further
"enquired into."

This groping after truth marks a point in the history of the knowledge of Dysentery on which little light was shed, from the point of view of etiology, till 1859, when Lambl observed Amoebae in the motions of a child suffering from Diarrhoea. The causal relationship of Amoebae to Dysentery was not definitely discovered till Loesch in 1875 described the case of a man who had suffered with Dysentery for three years; and at the post-mortem he found abscesses in the wall of the

gut swarming with Amoebae. He also found them in the faeces and injected fresh faeces containing those amoebae into the rectum of four dogs. In one Dysenteric symptoms were produced. Three weeks later the dog was killed and an ulcerated condition of the bowel was found. This parasite he named *Amoeba coli*. Further light was thrown on the subject by Koch and Kartulis working in Egypt, who observed the presence of Amoebae in Epidemic Dysentery in Egypt, demonstrating the presence of amoebae in Liver abscesses, and further establishing the causal relationship between Amoebae and Dysentery. Soon after Osler in America also discovered Amoebae in a liver abscess. His pupils Councilman and Lafleur in America, coincidentally with Lutz in Germany in 1891 definitely stated there was an Amoebic Dysentery and a Bacillary one whose type could not be defined. To Shiga in Japan belongs the honour of making the discovery of a specific *Bacillus* of Dysentery. This was in the year 1898, in the course of a severe epidemic in Japan, which date is accordingly a very important one in the history of Dysentery. Other observers confirmed this, notably Kruse in Germany during an epidemic in 1900. During this year also Flexner in the Philippines discovered a bacillus differing from Shiga's in certain sugar reactions. The pathogenicity of this variety was proved by an experiment on a prisoner sentenced

to death, in whom typical Dysentery developed and bacilli obtained in culture from the stool after swallowing a culture of the bacilli. Later Hiss and Russel discovered a bacillus, known as the 'Y' Bacillus, which has been associated with subsequent epidemics, and, especially, Asylum Dysentery. The subsequent history of Bacillary Dysentery is mainly one of isolation of different strains differing but slightly from the main types.

Meanwhile Schaudin in 1903, investigating the Amoebic question, differentiated *Entamoebae Coli* from *Entamoebae Histolytica* the pathogenic parasite. The subject seemed to be further complicated by Viereck's description in 1907 of a smaller Amoeba which he named *Entamoeba Tetragena*. In 1914 Walker and Selland published the results of their classical feeding experiments, proving that certain amoebae are cultivable and non-pathogenic; and that of the non-cultivable amoebae *Entamoeba Coli* can be parasitized on man, but is non-pathogenic; and that *Entamoebae Histolytica* can be parasitized on man and is pathogenic; and that *Entamoeba Histolytica* and *Entamoeba Tetragena* are just phases in the life history of the same organism, the latter being the phase prior to encystment of the parasite. Foremost among the observers of recent years on the protozoa inhabiting the intestinal tract of man is Wenyon of the Tropical

School of Medicine, London. The abundant material, provided during the past years of the war, has enabled investigators to confirm previous observations, and to appreciate that the present knowledge of the etiology of the Dysenteries is still very incomplete.

Classification./

Classification.

The main types can at any rate be divided according to present-day authorities into :-

- I. Protozoal Dysenteries
- II. Bacillary Dysentery.

Clinical Dysentery.

In addition to these two main types there are still a number of conditions which are clinically indistinguishable from them. To this group might be applied the term Clinical Dysentery. It may be that, with more detailed examination, these cases might prove to be of Amoebic or Bacillary origin. Therefore from a Clinical standpoint it is expedient to consider them as potentially one or other of these. Excluded from this group are infections by parasitic worms, such as the Ascarides etc. In certain diseases, too, Dysenteric symptoms arise, which are secondary to the primary disease. It must be borne in mind, however, that mixed infections do occur, and that there may be a true Dysentery complicating the original disease.

I. Protozoal Dysenteries.

Among the Protozoal Dysenteries some authorities classify --

a Leishmanic type

a Laveranic type.

The Leishmanic type.

is associated with Kalar-Aza - a disease in which there is frequently a mixed infection with a Dysentery of recognized type. It is still uncertain whether the Parasite of the Disease is capable of producing Dysentery by itself or not.

The Laveranic type.

As this introduces the controversial point as to whether Malaria can cause Dysentery, the present will be a convenient time for considering this question.

This factor bulked largely in the Dysentery that was common in Salonica where Malaria was very prevalent. Indeed, the large majority of our Dysenteric patients had suffered from frequent relapses of malaria. As malarial subjects fall easy victims to Dysentery, it was a common occurrence to have a combination of Dysentery of proved type with a relapse of Malaria. There were, however, many cases of mild

Clinical Dysentery, of which the question might be asked, - Were they Malarial in origin?

Some very important observations on this question were made by Duncan Graham, working in the Toronto General Hospital, Salonica. The stools of Malarial patients passing blood and mucus were examined, and out of 66 cases *Bac. Dysenteriae* was isolated in 62. One case was noted of Diarrhoea with a considerable amount of blood. On microscopical examination there were no characters of Dysentery, and it was negative Bacteriologically. Furthermore one noticed that the two diseases interacted, the one on the other, especially, in the more chronic type of Dysentery. It was not an infrequent occurrence to notice a man, whose stool had been keeping satisfactory for some days, commence to pass mucus again. If he were subject to Malaria, he might have a severe relapse the same evening or following day. This I frequently observed; but whether the Malaria relapse provoked the Dysenteric symptoms, or vice versa, is a matter of conjecture. I am inclined to the view that the Dysenteric condition, by lowering the resistance of the individual, provoked a relapse of Malaria. From a therapeutic and prophylactic point of view it is preferable to consider those cases of blood and mucus in the stool of malarial subjects as Dysentery superadded to Malaria, rather than Malaria with Dysenteric symptoms.

As a result of the examination of such a very large number of stools of soldiers convalescent in this country, some interesting observations have been made on the frequency, or otherwise, of the various protozoa in the bowel. Certain of these are definitely considered non-pathogenic, while some are doubtful, and one is definitely pathogenic. Thus some authorities divide the Protozoal Dysenteries into --

1. Amoebic
2. Flagellate
3. Ciliate
4. Bilharzial.

For practical purposes the Amoebic type is by far the most important, and is the one which chiefly concerns us. The other groups will be briefly considered before passing to the important Amoebic group.

(2) Flagellates have frequently been described as occurring in the faeces. The most important member of this group is

Giardia (Lamblia) intestinalis.

In a series of over 2000 cases examined and reported on by the Medical Research Committee in 1917 there were 16.1 per cent of cases infected with *Lamblia*. This was a result of three examinations for each case. In the active stage the parasite inhabits the small intestine, and, becoming encysted, passes into the

large intestine. It is only in the presence of Diarrhoea that the active flagellae swarm in the stool and quickly perish. The encysted parasite can, however, be a source of infection by being swallowed with food.

The pathogenic role of this parasite is still a disputed point among authorities. There seems, however, to be a consensus of opinion as to their power to irritate and aggravate an already existing Dysenteric lesion. The infection is apt to be most persistent and seems to recur periodically in spite of all treatment. Thus cases of Amoebic Dysentery with combined Lamblia infection may be cured after Emetine treatment so far as the amoebic element is concerned, but the Lamblia persist.

(3) Ciliate.

There is one member of this group, the Balantidium Coli, which deserves mention. It is a large organism which lives in the large intestine. Its usual host is the pig, which passes it in an encysted state, and is thus swallowed by man. By actual invasion of the wall of the large intestine it produces Balantidial (Ciliate) Dysentery.

In the series of cases above quoted there was not a single case of infection with Balantidium.

(4) Bilharzial./

(4) Bilharzial.

There is a Bilharzial infection of the large intestine, but this is usually secondary to Bilharziosis of the urinary organs.

The disease is common in Egypt and is very chronic in type, and very resistant to treatment. It is recognised by the presence of the typical ova in the faeces.

(1) AMOEBIc DYSENTERY.A. Etiology.

In the historical resumé the evolution of our present knowledge of the life history of the *Entamoeba Histolytica* has been traced, so that there is now recognised -

1. Active Amoeboïd stage,- *Entamoeba Histolytica*.
2. Smaller amoebic preencysting stage,-
Entamoeba Minuta.
3. An encysted stage,-
Entamoeba Tetragena, or
Tetragenous cysts.

All other Amoebae found in the stools, including *Entamoeba Coli*, have been definitely proved non-pathogenic.

The following is a description of the active amoeboid *Entamoeba Histolytica*,--

"Its size is from 25-30 u. Shows active movements
"with protrusion of pseudopodia. The ectoplasm
"is well defined from Endoplasm which is fairly
"homogeneous and may be vacuolated and frequently
"contains red blood corpuscles. The nucleus has
"a thin limiting membrane; Chromatin is arranged
"peripherally and is sometimes massed in clumps.
"At the centre of the nucleus a granule (Karyosome)
"can be distinguished. The nucleus is generally
"placed eccentrically."

This active *Entamoeba* is passed in the stools of an

acute case and is very perishable.

The next stage, "*Entamoeba minuta*" is observed in the stools when they may be diarrhoeal or approaching normal. It is considered to be a pre-encysting stage. It measures from 10-20 u. There is no definite ectoplasm; the endoplasm is vacuolated, Chromidia are frequently seen in the cytoplasm. The nucleus is small and rich in Chromatin.

The Encysted stage is voided in formed stools and is present usually between attacks. "It is a "spherical greenish refractile body, 10-14 u., enclosing a single mass of cytoplasm containing four "nuclei which are often in pairs at opposite poles "of the cyst. They also frequently contain one or "more rods of a homogeneous highly refractile substance called 'Chromidial bodies'. There may be "single vacuoles in the cytoplasm."

The causal relationship of the *Entamoeba Histolytica* to Dysentery is proved by feeding experiments, and, also, by findings at post-mortems, when the parasite can be discovered in abundance in the sub-mucous coat of the ulcerated large intestine and also in the pus of liver abscesses. By feeding animals with the parasites thus isolated characteristic Dysenteric lesions are re-produced. The following are the very significant conclusions drawn by Walker and Sellards from their feeding experiment

on twenty volunteers with Ent. Histolytica:-

1. Non-cultivable.
2. Microscopically Ent. Histolytica after short incubation period appeared in stools of every parasitized man and persisted for an indefinite time..
3. 17 parasitized after first feeding.
 - 1 ,, after third feeding.
 - 2 kept as controls after first feeding.
4. Incubation period 1-44 days: average 9.
5. Possible to obtain -
 - (a) Encysted E. Tetragena exclusively in stool of those ingesting E. Histolytica only;
 - (b) Mobile E. Histolytica only in those who had ingested Tetragenous cysts only.
 - (c) Alternation of Tetragena cysts and mobile E. Hist. in the stools of a man who had ingested Tetragenous cysts only.

This alternation was coincident with attacks of Dysentery in which the mobile forms were passed.

Further

6. Of the 18 men parasitized with E. Hist. 4 or 22.2% have developed Dysentery.
7. Incubation period 20, 95, 87, 57 days: average 64-8.
8. No case of Dysentery from ingesting Entamoeba Histolytica from an acute case or liver abscess.
9. All experimental Dysenteries resulted from ingestion of Tetragena cysts from the normal stool of a "carrier".

Mobile Entamoeba Histolytica from an acute case generally die and disintegrate in the juice of the stomach.

Thus we see that the important etiological factor in the spread of Amoebic Dysentery is the "cyst" form of the parasite. Accordingly, whatever conditions are favourable for the preservation of its life assist in the dissemination of the disease. For example,--

Moisture is one of those favourable conditions; hence the "cyst" thrives best in a warm humid atmosphere.

The ideal nidus is the soft stool of a "cyst carrier" on damp ground. Where the soil becomes polluted in the neighbourhood of water supplies, such as surface wells, the water gets contaminated and is a most fertile source of infection.

The common house-fly is another very important factor. Wenyon and O'Connor in Alexandria proved this, by catching flies in different parts of the town, and, on examining their dejecta finding cysts of *Entamoeba Histolytica* and *Entamoeba Coli* which were still alive.

Furthermore, they found that flies begin to excrete within half an hour of ingestion of faeces, and within twenty-four hours the whole is excreted. As a fly generally defaecates when it ingests food, it can thus, if it is already infected, contaminate further supplies of food and so very materially spread the disease.

Having thus gained an entrance to the stomach, whether it be by food or drink, the "cyst" is carried on in the course of digestion into the small intestine where it meets the Pancreatic juice. This dissolves the cyst wall allowing the young amoebae, which have developed from the various nuclei, to find their way into the large intestine, where they may, or may not, localize themselves and infect the individual. If they do not infect, they are passed in the faeces where, as living amoebae, they quickly perish. On the other hand, if a case is going to be infected, they find their way into and between Lieberkuhn's crypts to the submucous layers, where they commence their destructive work.

1. AMOEBIC DYSENTERY.

B. Morbid Anatomy and Pathology.

The description of earlier writers regarding the morbid anatomy of Amoebic Dysentery are very confused. The explanation of this is the fact that for a long time the two distinct types, namely, Amoebic and Bacillary, were not known as such. The most exact differential account of the early changes in the bowel in the two types is to be found in the work of Leonard Rogers. A further difficulty that obscures certain cases is the fact of there being a mixed infection so that, if the patient dies after a prolonged illness, there are advanced changes in the bowel produced by the two different diseases.

Rogers, in his long experience of the disease as complicated by Liver Abscess in India, has been particularly fortunate in being able to investigate the early change in the bowel in cases which have died of this complication. In such cases the Amoebic disease in the bowel may be latent.

Microscopically The primary lesion is an exudation into the submucous coat of the large intestine. Secondary to this there is congestion or haemorrhage in to the overlying mucous membrane. In the centre there is necrosis of the columnar epithelium and the yellow exudation underneath is visible. There is

an extensive small-celled infiltration in the sub-mucous layer and between the columnar epithelium. This is the commencement of the typical ulcer. Numerous Amoebae are also visible in a similar section in the submucous layer. It is noteworthy in this section that the glandular epithelium is intact almost up to the commencement of the ulcer. Extension of the infiltration along the transverse axis of the bowel in the submucous layer interferes with the circulation, and causes further necrosis of the epithelial layers. As the disease advances the muscular layers become invaded, and the subserous layer may be reached setting up a localized peritonitis, and in advanced cases sometimes perforation may occur. The picture may be complicated by invasion of the original ulcer with other microorganisms. The parasites, which may be seen invading the small blood vessels in the sub-mucous layer of the intestine, may gain access to the liver through the portal circulation. In this way the Amoebic Liver Abscess develops as a complication.

The naked eye appearances of these changes in the wall of the large gut, in the earliest stage, are manifested by small red raised spots, in some of which a yellow centre is visible due to the commencing ulceration. The marked elevation of the patches from the surrounding normal mucous membrane is a characteristic feature. Definite oval-shaped ulcers

in the transverse axis of the gut may be visible.

Rogers further illustrates the advanced changes in acute cases by an excellent plate, in which are noticeable the raised yellow ulcerated patches, some with black sloughs of irregular shape, and particularly noticeable are the extensive patches of healthy mucous membrane between the diseased portions.

The earliest changes occur in the caecum. It is noteworthy that the Ileum is not invaded in pure Amoebic Dysentery. The Appendix, however, may be affected.

In the healing or Chronic stages of the disease may be seen smooth-floored ulcers depressed and free from the "gelatinous infiltration of the early stage". Finally, they leave pigmented scars; while between these healed ulcers there may be perfectly healthy mucous membrane.

In an advanced case it may be possible to see all those changes at different parts of the gut, thus proving the relapsing nature of the disease, with its acute exacerbations coming on during its chronic course. Rogers has further drawn attention to the marked limitation of the disease to the upper part of the large gut, and especially the Caecum, in those cases dying of Abscess of the Liver, where past evidence of the disease may be,

merely, the presence of several healed pigmented ulcers. In some of those cases there may be healing chronic ulcers, scrapings from the floor of which reveals the Amoebae. In 98% of fifty post-mortems in cases of Amoebic Abscess Rogers found evidence of either active or previous amoebic ulceration in the bowel. Thus he has demonstrated the very close relationship between the two conditions.

Having now considered the Etiology and Morbid Anatomy of Amoebic Dysentery, which represents the main type of the Protozoal Dysenteries, we shall proceed to the discussion of the Etiology and Morbid Anatomy of Bacillary Dysentery.

II. BACILLARY DYSENTERY.

A. Etiology.

The Bacillus isolated by Shiga originally earned its title of being causative of Dysentery by being found in the faeces of those suffering from the symptoms of Dysentery, during an epidemic raging in Japan. At the post-mortem there were structural changes in the large intestine and scrapings from the ulcer revealed the bacillus. Experimentally the disease was reproduced in animals by inoculating them with the bacilli.

Furthermore, it showed specific agglutinating action with the blood of those suffering from the disease. Subsequent researches have still further substantiated the important rôle that Shiga's Bacillus plays in the disease, and have demonstrated that there are two main groups of Pathogenic Bacilli with certain sub-groups.

1. B. Shiga - Mannite non-fermenter.

2. B. Flexner - Mannite fermenter.

The Bacilli present certain characters in common and are diagnosed by the following methods and tests:-

1. Microscopically, they are short thick rods, 1-3u. long and with rounded ends. Most authorities seem agreed that they are non-mobile, but display vigorous Brownian movements. They stain well with

aniline dyes and are decolorized by Gram's method.

2. Culturally.

They grow well on the ordinary media - Agar-Agar, gelatin, bouillon and milk.

By prolonged sub-culturing, especially under different conditions, certain characteristics may be lost and others acquired. This applies more especially to their power of fermenting certain sugars with acid production.

3. Sugar reaction.

This is the important test for differentiating the various Dysenteric Bacilli from one another. The tendency is to multiply various types, in accordance with slight differences in those reactions.

Acid production in Glucose is a property common to all the Dysentery Bacilli.

Mannite is the important sugar for differentiating the main types into --

B. Shiga or Mannite non-fermenter.

B. Flexner or Mannite fermenter.

By their reaction with other sugars such as Maltose, Dextrose, Saccharose, the Flexner or Mannite fermenters are subdivided into various sub-groups, such as Strong, Flexner V, etc. The Dysentery Bacilli are distinguished from the Coli group by being non-

lactose fermenters.

It has been observed by Bahr that passage through the intestinal tract of the fly may exert a temporary influence on their powers of fermentation, but the one reaction that remains constant is that of Shiga and Flexner Bacilli to mannite and dextrose.

4. Agglutination.

This test is another valuable one for diagnosing the Dysentery Bacilli in conjunction with the Biochemical. It depends on the formation of agglutinins in the blood of the person suffering from the disease, so that, when that blood is tested with Laboratory strains of Bacilli of known nature, the bacillus may be identified. As this reaction does not usually appear before the 7th day and is sometimes delayed till the 21st, it is not of use as a diagnostic means early in the disease. It is possible, however, to get a very powerful agglutinative serum by inoculating rabbits with killed cultures of Dysentery Bacilli or their toxins. A potency of 1 - 1500 can be got or some even claim 1 - 20,000. If different varieties of the Bacillus are used and a series of sera thus obtained, it is possible by testing the unknown suspected Bacillus against a series of such to determine its nature if it be a Dysentery one.

There is a further great field for the use of

agglutination reactions in the convalescent to ascertain previous disease and thus diagnose the case retrospectively so to speak.

On account of certain limitations and apparently variable results of agglutination tests, there is considerable doubt among some observers as to its value.

There has been a great deal of work done on agglutinations as diagnostic of Dysentery, more especially, during the last few years. Some very interesting facts are set forth in the Medical Research Committee reports, which emphasize the necessity of ascertaining the standard for diagnosis. In order to do this it is necessary to know the upper limit of agglutinations in normal healthy individuals, supposed to be free of the disease or previous history of it. For this purpose the blood of 792 normal persons was tested with standard strains of Bacilli, with the following results:-

Bac. Shiga in a dilution 1-32 : 30% were positive
but 1-64 barely 1%

Accordingly complete agglutination in dilution 1-64 within 2 hours at 37°C may be considered a diagnostic.

In the case of B. Flexner it was a more unreliable test, but it was considered that complete agglutination within 2 hours at 37°C in a dilution 1-256 may be accepted as diagnostic.

The conclusion drawn in the Report is,--

1. Every positive test to B. Shiga in a dilution of 1-64 or above indicates a previous infection with Dysentery Bacilli.

2. A negative serological test does not exclude infection. This was proved by only finding ten positive agglutinations out of nineteen convalescents who were excreting B. Shiga in the stools.

The estimate is that 60% of Chronic Dysenteries or convalescents, who are still passing Bac. Shiga in the faeces, show a positive agglutination and in the case of Flexner infection 20%.

In the acute stage of the disease it has been observed in Shiga infections that agglutination is slow in appearing in the early days, but in the second or third week it is nearly always positive. This observation was made by Romm and Balashoff in Kieff during an epidemic when stools of a large series of patients were examined daily.

Persistence of Agglutinins in Dysenteric Infection.

Figures vary, but the Medical Research Committee found 21% are positive twenty-one weeks and over from the onset. Cases have been reported where there has been a positive agglutination ten months after onset of disease.

Coagglutination.

There is definite evidence, too, that both types of Dysentery Bac. may be agglutinated by the same case. This was noted in 3% of cases. In some cases there is undoubted mixed infection, but in others there is a "coagglutination". This seems to be more particularly the case with Flexner infections.

Another important consideration is the question as to how far the titre of agglutination will be raised by presence of Typhoid or paratyphoid infection or inoculation with T.A.B.

The evidence seems to point to the conclusion that on rare occasions an enteric infection may influence Dysentery agglutinations, but that as a rule they are unaffected.

By an examination of 65 healthy recruits, 32 of whom had been inoculated recently, and 33 had not, it has been demonstrated that T.A.B. vaccine has no effect upon natural Dysentery agglutinins. This is an important result when one considers that the very large proportion of Dysenterics, who have come under treatment in recent years, have been inoculated with T.A.B.

Widal Chart.

The value of the agglutination test is much enhanced by repeating the test in doubtful cases at

intervals of a few days beginning in the second week, and noting if there is a rising titre of agglutinins. Thus a Widal chart may be kept.

It will be seen, therefore, that the value of agglutination tests in assisting the diagnosis of Dysentery is considerable, provided the methods and strains of bacilli employed are standardized by controls.

In order, however, to identify the bacilli or possible new strain it is necessary to consider all the essential characteristics, such as morphology, Cultural reactions noting proteolytic action as tested on gelatine, indol production, fermentation reaction with sugars, and pathogeniety as well as serological test.

The importance of recognising different strains of Bacilli is recognised therapeutically in the preparation of Polyvalent sera. Thus Shiga recognises five distinct strains for the preparation of his specific serum, while others recognise more.

After considering the results of examination of several thousand cases as published by the Medical Research Committee reports, one realizes that there are many strains of Bacilli which are, with our present knowledge, a doubtful causal factor in the disease. As has been pointed out by Magner, many of those bacteria, though usually only saprophytic,

in some cases may assume a pathogenic role as secondary invaders. Some of those may be found in the faeces in conjunction with true Dysentery Bacilli or Amoebic cysts at times.

Sites where Bacilli are found.

Dysentery Bacilli, according to early observers, were believed to be localised to the intestines and lymphatic glands, but later observations have demonstrated their presence in liver, gall bladder and spleen after death. More recently they have been demonstrated during life in the blood and urine.

In Blood. Thus Darling (1912) discovered Shiga Bacilli in the blood of a person who died 4 days after-wards, and the disease was confirmed at the post-mortem. Ghon and Roman (1915) found Flexner Y Bacilli in the blood of 9 cases. These positive results were only obtained at the first examination early in the disease. Subsequent examinations a week later were negative in those cases. It may be that, if blood cultures be made more frequently early in the disease, the bacilli may be more frequently discovered. Here accordingly, as pointed out by the Research Committee Report, is a valuable and more reliable means of studying the various strains of Bacilli, and of determining which are true Dysentery Bacilli. Furthermore, it would make it possible

to have sera prepared from bacilli isolated during life from the blood.

In Urine.

Regarding the presence of Bacilli during life in the urine, the few times they have been discovered seem to have been during the acute stage. Many examinations made among convalescents proved negative or doubtfully positive.

Vitality of Bacilli.

This is a very important consideration, especially, from the point of view of prophylaxis. The behaviour of Bacilli, accordingly, under the following conditions are worthy of notice:-

(1) Temperature effects show interesting and unexpected results. Thus their vitality seems to be increased in water at freezing point, as compared with ordinary room temperature. Boiling kills them in a few minutes; and a temperature of 58°C for one hour kills them.

(2) Sunlight, according to Vincent and Muritat, destroys the Bacilli in 2-2½ hours. Shiga claims that 30 minutes' exposure kills them. The different estimates are no doubt due to greater strength of sun's rays in Japan. In the diffused light of a laboratory they live 8 days and in darkness 14 days.

(3) Soil has power of preserving Bacilli in winter and in a rainy season. Bacilli can be recovered from dejecta buried in soil for from 30-90 days. On linen folded up they last more than 30 days.

(4) Water. Vincent has made some very elaborate and careful investigation, and has demonstrated that cultures of Shiga bacilli emulsified with sterile water at 38° C survived 4-5 days and at 1° C 9-18 days. Further that, in water contaminated with other bacteria, their life is much shorter. He found, for example, Staphylococcus pyogenes, Bac. Coli, Pyocyanus, etc. highly antagonistic.

In ice and in darkness they have survived 41-68 days.

These etiological factors - cold, humidity and darkness - are more associated with temperate climates, but in warmer climates there is another factor, namely, the Fly, which plays an important part in the spread of the disease.

The Fly. Vincent experimented by feeding flies on cultures of Bac. Dys. under sterilized Bell glasses. Bacilli were found being excreted by them 4 days afterwards. Bahr isolated Shiga Bac. from intestinal tract of flies found on bed of patient suffering from acute attack of Dysentery.

The vitality of the Bacilli in the human faeces

after they are passed is low. It is stated that they die off in two days under ordinary conditions, owing to the antagonistic action of other bacteria of decomposition. Furthermore, bacilli in scrapings from ulcers in the intestine which lie deeper and are free from faecal matter are found to live longer than those taken from the faeces in the same case.

Persistence of Excretion of Bacilli ?

This is a very important question and it underlies the whole problem of the "carrier". Considerable light is thrown on this problem by the Medical Research Committee Report on the investigation of 878 cases Bacillary Enteritis invalided from the Eastern Mediterranean!

B. Shiga was recovered from the stools of 19 cases. In the majority of these cases more than four months, and in some cases six months, had elapsed since the onset of the disease.

B. Flexner was found in the stools of 9 cases, in some of which twenty weeks had elapsed since the onset. This makes a total of nearly 3% positive Bacillary findings in the stools, in contradistinction to - 31% positive Shiga agglutination

11.1% positive Flexner do.

It must be remembered, however, in this connection that these findings are from a type of case which is

essentially chronic. This series must be considered as a concentrated residue of troublesome cases from many thousands of cases running an ordinary course.

In the average acute case the Bacilli usually disappear from the stool fairly soon. Seligman, who investigated a large number of acute cases of Dysentery found Dysentery Bacilli in the stools of 38%. For each week of the disease his figures were

1st week	-	70%.
2nd	,,	53%.
3rd	,,	18%.
4th	,,	0

Experimental Dysentery.

Strong and Musgrave, in their famous experiment of feeding a negro with Dysenteric Bacilli, preceded it with Sod. Bicarb. to neutralise the acid of the stomach. Characteristic Dysentery was produced.

In animals fed by the mouth characteristic lesions do not appear, though Diarrhoea with mucous appears and Bacilli are recovered from the stool. Almost all animals respond to subcutaneous injections of cultures either dead or living. The rabbit is particularly sensitive and characteristic Dysenteric lesions are produced by subcutaneous injection. Further experiments show that a similar action is brought about by filtrates of young cultures of Shiga Bacilli. This toxin or endotoxin is comparatively stable, soluble and destroyed by heating 80°

after one hour. At room temperature it is potent after $4\frac{1}{2}$ months.

The rabbit and horse are very sensitive to it, whereas the guinea pig, mouse and monkey are hardly affected.

For example, the dose representing 50 minimum lethal doses for large rabbit causes only slight local symptoms in a guinea pig.

Feeding animals with the toxin or cultures of Bacilli by the mouth has no appreciable visible effect on the intestines; accordingly Sweet and Flexner concluded, that it was eliminative in character, and that intestinal change occurs only when the toxin reaches the intestine through the circulation. They found, moreover, that a Biliary fistula in a rabbit prevented the intestinal lesions, although the rabbit succumbs to the poison on the nervous system.

Their hypothesis is, that the poison is eliminated by the bile into the intestines, is reabsorbed in the small intestine, and has its selective action on re-elimination by the caecum and large intestine. A similar sequence, they conclude, probably occurs in man. The effect of the toxin on rabbits shows itself in two ways:-

1. On the large intestine causing Dysenteric lesion.
2. On nervous system causing paralytic symptoms and death.

Accordingly, it is considered by Horini, that there are

several toxins present which have a similar action in man.

Thus we see the essential difference between Amoebic Dysentery, where the results are consequent upon the direct action of the amoebae on the cells in their neighbourhood, and Bacillary Dysentery, where the action is presumably in the main, if not entirely, the result of the action of Toxins.

It has never been shown that *Entamoeba* produce toxin, or that they are capable of exerting any poisonous effect other than that of proteolysis of cells in their neighbourhood.

II. BACILLARY DYSENTERY.

B. Morbid Anatomy and Pathology.

(1) In acute cases in the early stages there is an intense congestion of the mucous membrane throughout most, if not the whole, of the large intestine and sometimes the lower few feet of the ileum.

Microscopical section at this stage reveals the fibrinous exudate into the mucous membrane and degenerating appearance of the glandular epithelium. There is oedema and possibly some haemorrhage into the submucous coat. The muscular coat is intact and there may be some oedema of the serous coat.

Macroscopically: Patches of greenish appearance show up against the general swollen mucous membrane. Extensive ulceration may occur, the ulcers being shallow and serpiginous, and the intervening mucous membrane oedematous. The ulceration does not as a rule go deeper than the muscular coat, and in this way differs from the ulceration in Amoebic Dysentery which tends to be much deeper.

Such are the changes that occur in a very acute case, but there may be all gradations varying from an early congestion of the mucous membrane up to an extensive ulceration with separation of dark gangrenous sloughs.

(2) Chronic case.

In the chronic case, which represents most frequently one that has commenced acutely and become chronic in nature, one sees in some places the scars of healed ulcers, and in other places many shallow depressed serpiginous-looking ulcers. The swollen affected portion of the bowel contains many of these ulcers. It is usually the lower half of the bowel that is affected with chronic ulceration.

Microscopically. The Lieberkuhn crypts may have entirely disappeared, the floor of the ulcer being formed of granulation tissue.

Thus after a severe attack there may be extensive destruction of the mucous membrane with formation of granulation tissue and some cicatricial contraction of the lumen. There may be thickening of the walls with some adhesion between the gut. Actual perforation is rare.

CLINICAL ACCOUNT of the DYSENTERIES.

In discussing the etiology and pathology of Dysentery there is such a marked difference between the two great types, that it was desirable to deal with each separately. The clinical picture, however, of the two cannot be so distinctly defined as separate entities. Accordingly, it seems more satisfactory to consider certain clinical types, and allude to special features which are more characteristic of one or other of the Dysenteries.

There are, no doubt, certain features which might enable one to make a fair guess at the diagnosis, but, without the assistance of the Bacteriologist, it is impossible to make an accurate diagnosis of the etiological factor. In Salonica both the Amoebic and Bacillary type occurred.

The Amoebic element was, to a large measure, introduced among the troops by theth Division, which had been at the Dardanelles and in Egypt in 1915 and came to Salonica in the autumn of the same year. Many other troops also came from Egypt where Amoebic Dysentery is common. In later years of the war Amoebic Dysentery was becoming much more prevalent, as a result of a spread of the infection among the troops so introduced. This knowledge of a man having previously been in Egypt made one much more

suspicious of the case being Amoebic.

Duncan Graham in a series of 2000 cases at the Toronto General Hospital ^{Salonica} up to early in 1917, gives the figures for Amoebic as 3%. Both Shiga and Flexner types of Bacillary Dysentery were common, he states.

I have not figures for the Hospital in which I worked, but we had a fair number of Amoebic, Shiga and Flexner types. There were two distinct periods in which my observations were made:-

1. November 1917 - end of March 1918.
2. End of June - end of November 1918.

During the first period the General Hospital was first formed into a special Dysentery Hospital for receiving the Dysentery cases already collected in individual hospitals. During this period, too, there was some difficulty in getting cases invalided out of the country, owing to enemy action at sea. Consequently, the chronic type of case had to be kept in Hospital in the country much longer than would otherwise have been the case. This, however, gave the Bacteriologist further opportunities for examining the stools of such chronic cases, and, in this way, a larger number of positive findings were possible, especially, among the Amoebic.

During the second period one saw a much larger number of the acute types of the disease, many of the

cases being severe Shiga's and Flexners. Some of the Flexner's were just as acute as the Shiga's, which experience is contrary to what is so commonly asserted namely, that the Flexner is a non-toxic type. A certain number of the Acute cases proved to be a mixed infection of Amoebic and Bacillary. Indeed, in more than one acute case there was a triple infection - Amoebic, Bac. Shiga, and B. Flexner.

During September the Influenza epidemic spread over our Hospital and very materially increased the mortality, which otherwise was fairly low. The virulent Influenza infection, with its attendant Broncho-pneumonia complication, found the patient, already weakened by Acute Dysentery and Malaria, an easy prey to its ravages.

Malaria was another disease which was apt to vary the Clinical picture of Dysentery, and frequently it very much complicated the diagnosis in acute cases. Such a large number of the patients had a bad Malarial history, that it was frequently difficult to be able to assign a correct explanation of certain pyrexias, even though blood films proved negative; for a negative blood film did not necessarily exclude malaria. On an accurate diagnosis in this respect depended rational treatment; and lack of due recognition of this malarial element might make all the difference to the ultimate recovery

of the case. When an acute Bacillary Dysentery, especially, the Shiga type was complicated with a severe Subtertian Malaria the prognosis was extremely bad.

During this second period it was possible to invalid out of the country a much larger number of chronic and debilitated cases, so that this group was not so long under observation as during the previous period.

A few preliminary remarks on the division of the cases in the Hospital in which I worked will explain how my experience in Amoebic Dysentery was limited to a certain type of case.

Cases on admission with a distinct history of having passed blood and mucus in the stools were sent to the Bacillary lines. The stools were soon examined; and, if the case proved to be Amoebic, was transferred to the special Amoebic part of the Hospital. Latterly there was a Clinical Division and a Bacillary Division, so that all cases, on admission as clinical cases, were first of all sent to the Clinical side, and, as soon as the type was diagnosed, the case was sent to the particular ward allocated for that type. As I worked entirely on the Bacillary and Clinical lines, I only had the opportunity of observing Amoebic cases up to the point at which they were diagnosed. As it sometimes, however, required

repeated examinations before a positive could be got, such cases might be under my care for a longer time. Moreover, in certain cases it might prove impossible for the Bacteriologist to diagnose the type, although potentially it might be an Amoebic. With these preliminary remarks I shall proceed to discuss, in the first place, the Clinical types of the Dysenteries and later the complications.

"Dysentery has been aptly described as a 'symptom complex' which indicates ulcerative colitis, 'acute or chronic, which may be due to various pathogenic agents and etiologically quite distinct."

This "symptom complex" includes:-

1. Abdominal pain.
2. Tenesmus.
3. Characteristic stools.

1. Abdominal pain: may assume a most severe form described as tormina. Indeed, this symptom is so characteristic of an acute case, that Dysentery was called by that name by the Romans. It is a very severe **gripping** or **twisting pain** which may involve the entire length of the colon. It may be excited by taking of nourishment or may come on spontaneously. It may be transient and be relieved by an evacuation of the bowel, or may persist quite independently for a time.

There may be tenderness over the colon, either along its whole length, or at localized parts. There may be also a certain amount of swelling at certain parts of the abdomen owing to thickening of the bowel.

2. Tenesmus. This may be rectal or vesical and is a most distressing symptom. It is likewise so characteristic that the French originally called Dysentery Ténésme. It implies a constant desire and straining to have the bowels moved. Perhaps the result may only be the passage of a little blood or mucus followed by a further straining. So exacting a feature may this be, that a severe case may be almost "glued" to the bed-pan. As Manson well puts it, "The nearer to the rectum the lesions, the more urgent the tenesmus; the nearer to the caecum, the more urgent the griping."

3. Character of Stools. The characteristic of the early acute Dysenteric stool is the presence of blood. This was the esalient feature that seemed to appeal to the ancient English authorities who called Dysentery "the bloody flux".

The mucus may be transparent, whitish and diffluent, or it may be more membranous, or even flaky. It may be merely streaked with blood, or

may be intimately mixed with it, or the blood may preponderate. Sometimes there may be large black clots where there has been ulceration high up. Later, as improvement occurs, the stool may be more fluid with bile staining and commencing faecal character. On the other hand, if the Dysenteric process is extending and gangrenous ulceration occurring, there may be large foul-smelling sloughs. In the commencing stages of ulceration the stools become mucopurulent, and later a considerable amount of purulent matter with blood staining may be passed. Thus it will be realised how the condition of the stool may reveal the progress of the case, and how very important it is to examine the stools daily in a case of Dysentery. This is the keynote of successful management of a case.

The appearance of a particular stool does not throw much light "per se" on the diagnosis of the type of Dysentery, though some consider certain features pathognomonic. But what is a great help in diagnosis is the observation of a stool, which one day may be fairly normal, and perhaps the next day blood and mucus may be present which may rapidly clear up only to reappear in a few days. Such a varying appearance is very suggestive of an Amoebic cause.

There is another type of stool which suggests

a Dysenteric origin as distinguished from a Chronic Diarrhoea of simple origin; namely, a fluid faecal stool in which flakes of mucus are suspended.

A stool, which is of bad omen, is one where mucus floats in a serous liquid reddish-brown or black with a foul smell. This is suggestive of gangrene.

Frequency.

The frequency of stools depends on the type of the disease, and may vary from four or five in a mild up to fifty or more in a severe case. There may be complete incontinence due to paralysis of the sphincter ani.

In an acute case the chances of finding the parasite or amoebae are much increased by obtaining blood-stained mucus for the Bacteriologist, for it is there that the Bacilli and mobile Entamoeba Histolytica abound. In the chronic case it is the Tetragenus cyst that is found, and this may occur in a normal or soft stool.

Even with Bacilli in Convalescents it has been observed, that they are discovered equally frequently in soft or diarrhoeal stools apart from mucus.

The Blood.

The Blood changes are of some assistance in determining the clinical nature of the disease, and

especially, in the diagnosis of certain complications.

In Rogers' studies of the blood changes he finds, on analyzing 60 cases of uncomplicated Amoebic Dysentery, that 75% show leucocytosis.

In 10, it was over 30,000.

In 17, it was 20-30,000.

Out of the 7 that had no leucocytosis 3 had Kali-azar which accounted for scanty white count. He considers that, when the count exceeds 26,000, the prognosis is grave. The differential count does not seem to be very helpful, except in presuppurative hepatitis and amoebic abscess of the liver where there is a marked leucocytosis with an increase of polymorpho nuclea cell, which is less in proportion to what one would expect in ordinary suppurative conditions.

In contrast to this, in uncomplicated Bacillary Dysentery a leucocytosis is rare, and, in cases where it does occur, it does not exceed 15,000.

In most of the cases under my observation the blood picture was liable to modification, on account of the frequent association with Malaria. In that disease it is stated by authorities, that during the pyrexial stages there is a leucocytosis, and in the apyrexia stage a leucopenia which, in some anaemic types of recurrent malaria, reaches relatively a marked degree. There is, however, a marked increase of the mononuclear cells in the differential count

and sometimes a marked Basophilia. Thus it will be seen that one could not expect to get the same assistance from a leucocyte count where the two diseases were combined.

Incubation.

Incubation period of the two types differs. In Bacillary Dysentery it was found to be 36 hours in feeding experiments. The usual is 3-6 days.

On the other hand, in Amoebic Dysentery it is much more variable, and apt to be much longer. This was proved in Walker & Sellard's feeding experiments, when the average period for parasitization to occur varied from 1 - 44 days: average 9; whereas the average period for Dysenteric symptoms to appear was 64.8 days in those who developed Dysentery.

Onset. In the Bacillary is usually quite sudden, whereas in Amoebic it is usually somewhat insidious, being ushered in by some days or weeks of diarrhoea and general indisposition.

Fever.

In Bacillary cases there is a temperature which is usually 101-102° at the outset and continues with remissions for several days. In ordinary cases it may be normal or only slightly raised thereafter. In severe cases where there is marked ulceration and mixed septic infection, there may be a swinging temperature for several weeks with an eventual recovery.

On the other hand, I have seen a severe Bacillary case with a temperature only very slightly raised after the initial rise.

In Amoebic cases apyrexia is a more marked feature, even at the onset, in uncomplicated cases. Later there may be pyrexia, as a result of mixed infection. In the course of a doubtful case, where the onset is apyrexial and there is later a slight rise to 99 or 100 each day and normal in the morning, this is suggestive of an Amoebic case complicated by Hepatitis.

The classification I propose adopting, in the Clinical description of the Dysenteries, is the following:-

I. Acute.

- (1) Severe or moderately severe.
- (2) Mild.

II. Chronic.

- (1) Following on Acute.
- (2) Insidious commencement.

I. Acute.

- (1) Severe or Moderately severe.

This is a more frequent clinical type in Bacillary than in Amoebic cases. The onset may be sudden with abdominal pain and the passage first of a faecal stool soon followed by mucus becoming blood stained. There is marked tenesmus, and the stools become very

frequent, and consist solely of pure blood-stained mucus. They may number fifty in the six hours, each being represented by just a little blood-stained mucus. The temperature may be raised to 102° or 103° . The pulse is relatively slow. The tongue is furred. The patient looks ill and has an anxious expression, the result of the tormina and tenesmus. There is the natural constitutional disturbance and disinclination for food. There may be considerable vesical tenesmus with scanty urine. Dysuria is sometimes a troublesome feature. In one case where there was retention I had to catheterize for ten days before the urine began to come naturally.

The abdomen is tender on palpation over the whole length of the colon from the caecum to the rectum. The tenderness may, however, be localised to the iliac fossae or splenic and hepatic flexures. The abdomen moves freely on respiration and there is no marked rigidity.

The temperature remains elevated with a remission of $1-2^{\circ}$ for the next few days, and about the 5th or 6th day may have gradually come down to normal. In a moderately severe case there might be only a slight rise of temperature in the evening for a week or so, coincident with the pathological process going on in the bowels. In a very severe case it is more common to see an irregular spikey temperature with a remission

of 1° or 2°. In one of my cases of Shiga Dysentery this continued for over four weeks with ultimate good recovery.

The clinical picture of this and other types is modified according to the routine treatment adopted.

The progress of the case is daily observed by the stools, which, in the type under discussion, may continue very frequent for 48 hours, but they begin gradually, in moderate cases, to become fluid in character with bile staining and still a good deal of mucus. After about five or six days, in moderate cases, they may be becoming diarrhoeal in character with some mucus mixed with it. In severe cases, where the ulcerative process is marked, there is a good deal of mucus. This type of case is apt to develop into the Chronic. I have seen cases, which, for nearly a month passed mucopus regularly, eventually clear up and make a good convalescence.

In very bad cases foul smelling sloughs begin to come away later on. This marks the gangrenous type, which is apt to be fatal.

The patient, as the disease progresses, gets very thin and the skin tends to get dry and withered.

After the first few days, in a case that is doing well, the tongue cleans and the patient is usually anxious for food which he is not able to have.

The tormina and tenesmus diminish rapidly under

treatment, but in severe cases the tenesmus continues and eventually lapses into complete incontinence.

In the later stages of a very severe case the very troublesome and intractable hiccough which develops is a very bad sign. Vomiting was not a common symptom in the cases I saw. Occasionally it occurred at the commencement of an acute case and sometimes was a bad late phenomenon.

There is one form of this severe case described which, however, did not occur among my cases, namely, the Choleraic. In this kind of case the onset is so acute and the loss of fluid so great, that it simulates Cholera in the collapsed algid state into which the patient speedily lapses. With prompt treatment such cases may recover, but they are usually fatal.

(2) Acute Mild type.

There was a very large number of our cases of this type. The onset may be acute; but in 48 hours the whole acute aspect may have gone and the patient rapidly becomes convalescent. For the first 48 hours the temperature may be up to 101° , with a fall next morning, and then up to 100° , and then down to normal.

The stools early become diarrhoeal in nature with flakes of mucus suspended in it. Indeed, fre-

quently, cases were admitted to Hospital in this state, when it was at times only the history of blood and mucus in the stools a few days previously that justified the diagnosis of Dysentery. Frequently those mild cases were of the Flexner type of Bacillary Dysentery, though I have seen Shiga cases equally mild. Owing, however, to the short duration of acute symptoms, which was frequently passed before admission, a large number of those cases remained as clinical, neither Amoebae nor Bacilli being discovered. Relapses occurred in a certain number of this type, and they might prove to be severe. I have seen cases admitted of this mild type which, during convalescence, developed a severe relapse in which Bac. Shiga was discovered, though in the first attack nothing was found. Such a case was obviously open to another interpretation, about which it would be impossible to dogmatize, namely, that it was a re-infection while in Hospital.

Another type of this mild relapsing case very commonly proved to be Amoebic in origin. It was on the whole more frequent to find *Entamoeba histolytica* in this kind of case than in the very acute type.

Although mild in nature, it was just as necessary to adopt rigorous care in their treatment, on account of the liability to complication.

This is the kind of mild case which was open to

the interpretation, Malarial Colitis, when occurring in malarial patients.

It has been previously stated how many of those, which were at one time suspected to be so-called Malarial Colitis, proved to be positive Dysentery which was frequently of Flexner type.

A certain number of this mild type of case developed into Chronics, on account of persistent mucus in the stools.

II. Chronic.

(1) Where previously Acute.

As has been remarked, an acute case may, instead of clearing up in the course of a few weeks to normal convalescence, continue to pass mucopus in the stool. Some may pass large quantities of this, while others may just have a little mucus occasionally appearing in the stool with slight diarrhoea.

The latter was the type in which one looked specially for Entamoebae Histolytica cysts. It was also not an uncommon sequence for mild Flexner cases to follow. A considerable number proved to be of this nature, which necessitated invaliding. There was latterly a recognised rule in the Salonica Command that any case, continuing to pass mucus after eight weeks, should be invalided.

The Chronic Shigas were generally rather of the

severe type with more mucopus. This Chronic case was apt to be rather deceptive as regards the character of the stool; for example, if the morning stool were seen, it might be semi-soft with a considerable amount of mucopus, while later in the day there might be a soft fairly normal stool. Chronic Bacillary Dysentery was very unsatisfactory, as regards its course, in comparison with the same kind in the Amoebic type, where rapid improvement in the local condition followed specific treatment.

(2) Insidious from the start.

It has been pointed out how the Acute Bacillary case usually started with an acute onset, or perhaps with a day or two of Diarrhoea, and that in the Amoebic case there may be some days or even weeks of irregular diarrhoea.

In others, which were usually of the Amoebic type, there was a history of chronic diarrhoea, perhaps four or five liquid faecal stools a day, lasting for six weeks or more. I have had such cases admitted to my wards, on account of a slight acute attack, with the passage of blood and mucus, arising during the period of this chronic diarrhoea. The patient looks thin and anaemic. There is no pyrexia. Indeed, the temperature may be subnormal. There may be some localized tenderness in the right or left

iliac fossa. At first the bacteriological reports may be negative. The acute blood and mucus may subside readily, but the chronic diarrhoea persists with or without mucus. Subsequently, it may be after one or more examinations, Tetragenous cysts are found in the stool.

Sometimes this Chronic Diarrhoeal case without any blood or mucus was admitted as N.Y.D. (not yet diagnosed) Diarrhoea. It might be in Hospital for five or six weeks and then be sent out to Convalescent camp temporarily cured, but be re-admitted in the course of a few months with a recurrence of diarrhoea. Such cases, after repeated examination, frequently proved to be positive Amoebics. This type of Chronic Dysentery is peculiarly characteristic of the Amoebic group.

There is, however, another kind which is sometimes called "latent", namely, the case which may show no symptoms of Dysentery whatsoever, but its nature is ascertained in virtue of a later complication, such as Amoebic Abscess of the Liver. Rogers has demonstrated this repeatedly during post-mortems on cases of liver abscess, where healed Amoebic abscesses are found in the caecum, or, it may be, a healing abscess from the floor of which *Entamoeba Histolytica* was discovered microscopically. The fact that the seat of the trouble is in the Caecum accounts

for the absence of the usual symptoms, especially if the ulcer be small.

I have seen one case, where there was occasional diarrhoea alternating with constipation and persistent tenderness in the right iliac fossa, so simulate Appendicitis, that operation was performed and a fairly normal appendix revealed. The following day *Entamoeba Histolytica* was discovered in the stool, though repeated examination previously had been negative. Case No. 11.

Such then are the usual clinical types of the diseases which are etiologically so different.

Complications of Dysentery.

Inasmuch as the morbid anatomy of the two different types of Dysentery is primarily an ulcerative Colitis, one does not expect to get very different different clinical pictures, so far as the ordinary course is concerned. In dealing with the complications, however, the conditions are somewhat altered; for, in the Amoebic type we are dealing with the localized action of a parasite, whereas, in the Bacillary, we are dealing with the effect of a toxin. Furthermore, the earlier pathological changes in the inner coat of the bowel may be indistinguishable clinically, but the more advanced changes tend to differ. Inasmuch as there is a greater tendency to the extension of these distinctive processes into the deeper layers of the intestine in the Amoebic type, there is, accordingly, the greater liability to different complications. Therefore, the very nature of a complication arising in the course of a Clinical Dysentery may be very strong presumptive evidence of the particular type of Dysentery whether it be Amoebic or Bacillary.

Hepatitis.

In this respect Hepatitis is a complication which is very suggestive of the case being Amoebic.

Much of our knowledge of this complication, in its relation to Dysentery, and in its being a presupplicative stage frequently of Amoebic Abscess of the Liver, we owe to Rogers who investigated this subject so thoroughly in India. He has established beyond dispute --

1. That an acute Hepatitis may occur in the course of an attack of Amoebic Dysentery.
2. That, where untreated by Emetine, this may develop into so-called Tropical Abscess or Amoebic Abscess of the Liver.
3. That there may be Hepatitis, where the Dysentery is latent, which yields speedily to specific Emetine treatment.
4. That Amoebic Abscess of the Liver is always associated with either active or latent Dysentery.
5. That in all cases of Amoebic Abscess, there is a history of presupplicative Hepatitis for periods varying from 9 days to over a month.

Since Rogers' earlier discoveries there has accumulated abundant evidence to attest to the wonderful effect of Emetine, in averting Tropical Abscess, by efficient early treatment. Hence the great importance of recognizing early the presupplicative stage of this serious complication. From the diagnostic point of view, he emphasizes the importance of a low remittent fever, accompanied by a marked leucocytosis with 70-80% polymorphonuclears. A recent observer states that in Amoebic Dysentery there is an eosinophilia 7-9% present, which, with

the onset of Hepatitis, disappears and is succeeded by Polymorphonuclear leucocytosis.

Hill, who has worked a good deal on this subject of Presuppurative Hepatitis, calls special attention to certain diagnostic features in patients from the Mediterranean area:-

1. Pain felt in the right shoulder and down the arm is very suggestive, as it may be reflexly caused by pain at the diaphragmatic surface of the liver.
2. Upward enlargement of the liver affects the relative dulness of the liver much more than the absolute, and, to detect this, one must compare the findings with the upper limit before the onset of Hepatitis. Accordingly in every case of Dysentery it is advisable to note this upper limit at the onset for future comparison.
3. Lumbago with fever is usually Amoebic Hepatitis.
4. Right basal pleurisy is usually Amoebic Hepatitis.

Great stress is laid on the value of X-ray for diagnosis, in that it clearly shows the upper limit of the diaphragm, and so any fixation of movement may be noted. The limitation of its use, however, in diagnosing actual abscess formation is considerable; for cases have been noted where, in spite of the fact that there was no difference in the density of the shadow of the liver, a large abscess has been present. Conversely, denser shadows have been noticed suggesting an abscess, and yet none have been found. This, of course, applies to the substance of the liver.

Any collection at the upper surface pressing up the diaphragm would be revealed, as the upper margin of the liver is very well shown by X-ray.

In acute hepatitis there is distinct liver tenderness and enlargement. The discomfort is such that the patient may have to lie on one side.

One marked case under my observation developed hepatitis in the course of a mild clinical Dysentery. There was a slight remittent fever rising to 99° or 100° in the evening. Previous examination of the stool had proved negative. The onset of this complication led to renewed examination of the stools being made, from one of which its Amoebic nature was proved. As the case was removed to the Amoebic Ward it was not under my care longer.

Hepatitis in Bacillary Dysentery may be said to be most rare. Rogers stated that in 125 Post-mortems he made in primary and secondary Dysentery, 38% of which were Bacillary in nature, he never met with a case of Portal Pyaemia or other serious disease of the liver resulting from Bacillary Dysentery.

Among our cases of Bacillary Dysentery there was liable to be Hepatitis of Malarial origin. This was of quite a mild type, which yielded to specific treatment and was never followed by suppuration.

Amoebic Abscess of the Liver.

In a certain number of cases of Hepatitis of the Liver, an abscess develops. The one condition merges into the other so insidiously, that the detection of pus is sometimes a most difficult matter. As the presuppurative stage is so important it has been dealt with at a greater length, and I propose only to deal briefly with the mature condition itself. Fortunately it is becoming very much rarer now, since cases of Amoebic Dysentery are submitted to early routine treatment with Emetine.

The abscesses may be multiple or single. Rogers' statistics show that 70% are single, and that, in fully 50% of the multiple, two abscesses are present. The multiple abscess are much more serious and tend to swell the deathrate from this complication.

The large single abscess occurs much more frequently in the right lobe, where they are apt to be very insidious in showing signs or symptoms, on account of the great thickness of the liver substance in the right lobe. They may attain to large size with a thick fibrous wall containing a quantity of pus.

The single abscess in the left lobe, occurring in 16.4% of Rogers' series of single abscesses, is usually detected much earlier, on account of the thin liver substance. As it is now amenable to early

surgical treatment, it is less fatal.

The suppurative stage may show itself in an increase of fever, 100° in the morning to 103° or 104° in the evening. In addition to this there may be very copious sweating, especially, in the acute cases. There is usually great prostration accompanied with wasting.

In some cases it may be the further onset of complications that lead to the detection of the abscess. Thus a not infrequent sequel before the days of Emetine, Rogers says, was the rupture of the abscess into the lungs, which resulted in a quantity of characteristic liver abscess pus - "anchovy sauce appearance" - being coughed up. When under observation in the presuppurative stage this would in all probability be suspected by physical signs at the right base, or by pain in the right shoulder as previously pointed out.

The abscess, on the other hand, may open into the pleural cavity causing signs of a pleural effusion, or into the stomach or bowels. A very serious complication is rupture into the peritoneal cavity with the onset of abdominal symptoms.

In some cases it may be necessary to resort to exploratory puncture of the liver to clear up the diagnosis, and localize the abscess.

ABSCESS OF THE BRAIN.

(1) In Amoebic Dysentery.

This is a very fatal complication and may not occur till some time after signs of Dysentery or Abscess of the Liver have disappeared. When it has occurred, the symptoms have been similar to ordinary cerebral abscess. A certain number of cases have been described and so it must be mentioned as a possible complication. I have seen a cerebral abscess secondary to middle ear disease developing in the course of Amoebic Dysentery, but I have never seen a true Amoebic abscess.

(2) In Bacillary Dysentery it has been remarked that cerebral symptoms developing are of toxic origin. I should like, however, to mention an interesting and unusual complication that occurred in one of my cases, as it might be considered potentially a possible abscess. It was a case of haemorrhage into the anterior part of the Caudate nucleus on the left side complicating a Bac. Shiga Dysentery. The patient died from pressure on the lateral ventricle. When one considers that Bacillaemia does occur occasionally - as, for example, when Shiga Bacillus was discovered in the blood four days before death - it is quite a possible conjecture that the bacilli might lodge in such a damaged spot when the blood clot would be a favourable nidus.

Parotitis.

This complication sometimes occurs. In Amoebic cases the parasite has been discovered in the gland. On the one occasion I saw this complication the case was a severe one of Dysentery clinically complicated by Malaria.

The onset was sudden and acute with Dyspnoea and swelling of the Parotid with cellulitis of the left side of the neck about the fourth week of the disease. The Dysenteric symptoms had been improving, but about this time there was a slight exacerbation of the dysentery. Suppuration ensued and free drainage was provided by an incision at the angle of the jaw. The Cellulitis gradually subsided, and the abscess of the parotid gland healed well. Later lung complications developed and the patient died some weeks later.

Haemorrhage.

(1) Intestinal.

This complication may occur in both Amoebic and Bacillary Dysentery.

When one considers the pathological processes at work in the ulcerative stages of Dysentery, it is not surprising that a severe and sometimes fatal haemorrhage occurs from the bowel.

Clinically one expects to see a certain amount

of blood in the stool in an ordinary acute case, but in some cases this becomes so excessive, as to merit the title of a complication of the disease.

(a) In Amoebic Dysentery with the larger, deeper type of ulcer, and the tendency for the process to burrow deeply, there is the greater liability to severe haemorrhage.

Sometimes the disease may be latent in the caecum till a severe haemorrhage leads to the diagnosis. In this connection I quote an interesting case of a severe intestinal haemorrhage 24 hours after an appendicetomy. A sample of the first lot of blood clot that was passed was sent for examination and was found to be swarming with *Entamoeba Histolytica*. It was wonderful in that case what a rapid recovery ensued with cessation of haemorrhage after Emetine treatment was commenced. -- See Case No. 11.

(b) Bacillary Dysentery.

In Bacillary Dysentery, on the other hand, the pathological condition is usually more superficial. Still, here also, especially in cases of mixed infection with other organisms in the intestine, there is apt sometimes to be severe haemorrhage. It is not necessarily a fatal complication. One case under my care of B. Shiga Dysentery had a very severe haemorrhage from the bowel in the fourth week of the disease. He had been running a septic "spikey"

temperature for some time before the haemorrhage and the stools had contained mucopus. After 24 hours there was no recurrence and he eventually made a good recovery.

In another case of very severe Shiga Dysentery there was serious haemorrhage about the third week. This continued to recur for several days, till it eventually cleared up under rigorous treatment. Dysenteric symptoms, however, continued, and about two weeks later, during the Influenza epidemic, he developed Broncho-pneumonia and died. At the post-mortem it was interesting to see the condition of the bowel, which in many places showed healing going on. There was not visible any gross lesion or ulcer to account for the haemorrhage.

In Bacillary cases there may be an amount of blood in the stool, almost amounting to a severe haemorrhage, the result of general oozing from the extensive superficial congested area.

(2) Haemorrhage in remote parts.

So far we have considered direct haemorrhage from the bowel, but there are indirect complications where haemorrhage occurs in other parts than the bowel secondary to - emboli, thrombosis, or endarteritis. In one case already quoted there was a haemorrhage into the anterior part of the Caudate nucleus on the left side, complicating a case of very

severe Shiga Dysentery. The strict localization of the haemorrhage to the anterior part is interesting inasmuch as it meant rupture of a blood vessel which was a branch of the anterior cerebral and not the more common artery of cerebral haemorrhage - the lenticular striata branch of the middle cerebral. The man was only 28 and had marked Arteriosclerosis but no Syphilitic history. There was a malarial element in the case, but no parasites were found in the blood during life nor post-mortem.* Case No. 10.

Perforation.

As a result of the invasion of the deeper coats of the bowel the serous coat may be involved and perforation may occur. This is rare in Bacillary disease, but in Amoebic Dysentery it occurs more frequently. This is as one would expect on contrasting the pathological processes of the two diseases.

Where this occurs in the course of an acute gangrenous dysentery the prognosis is extremely grave, because of the friable condition of the bowel, which would render surgical intervention useless.

It is remarkable how insensitive the peritoneum appears to be in case of leakage and general peritonitis in those advanced cases. Whether it is that the bowel is in a semi-paralysed and insensitive state is uncertain, but on examination of the abdomen the

walls seem more flaccid than one would expect to find in a case of perforative peritonitis.

In the more Chronic type of case there is the the liability to localised peritonitis, especially in the region of the caecum and ascending colon. This is likewise more frequent in Amoebic Dysentery. If perforation occurs, it is likely to be more localised and retrocolic in position. Such cases are apt to be mistaken for appendicitis.

I saw one case where a pelvic abscess developed in the course of a clinical dysentery. The case was operated on and a large collection of pus drained - enterotomy being performed at the same time to relieve the tendency to paresis. It was not advisable at the time to search for any leakage as the patient was too ill, but the presumption from the physical signs was, that it was somewhere about the sigmoid.

A few days after the operation he was doing well. I have not heard the subsequent history.

Appendicitis.

It has previously been pointed out how this may be simulated by localized pain and tenderness in the right iliac fossa caused by Amoebic Dysentery. Likewise in Bacillary Dysentery, especially Shiga, this may be a feature most difficult to diagnose at times from true Appendicitis. Suppurative Appendic-

itis may occur in the course of an ordinary Dysentery as a concomitant disease, but cases have also been reported directly due to *Entamoeba Histolytica* where the parasite was found in the suppurating appendix.

Oedema.

During convalescence this is sometimes a complication. It was a marked feature in two of my cases; in one there was extreme oedema of the lower limbs during convalescence from a moderately severe B. Shiga Dysentery. There was no evidence of thrombosis, nor albumen in the urine, nor of heart disease. The other case, also a Shiga Dysentery which had been moderately severe and had lapsed into a Chronic type, developed a general Anasarca. The face in particular looked so swollen, as to suggest renal disease. Yet there was no albumen in the urine.

Both cases were invalided by Hospital ship. There was every reason to expect they would make a good recovery.

Peripheral Neuritis.

I have not seen a definite case of this as a complication of Dysentery, but it is described as occurring. Thus one recent observer in Germany describes it as occurring in 1% of cases during convalescence.

In certain severe cases, where there was extreme emaciation, the patients might complain of symptoms suggestive of neuritis, but this could be explained by pressure effect on the sciatic nerve with lying.

When true peripheral neuritis does occur it is the result of the toxin of Bacillary Dysentery, analogous to the peripheral neuritis of Diphtheria.

There is one other complication to which I wish to make special reference, namely, Arthritis.

ARTHRITIS.

This is an interesting complication of Dysentery to which but scant reference is made in the literature of Dysentery, though it has been recognised as far back as the days of Hippocrates. As it occurred in certain of the cases under my care, I propose to discuss it rather more fully than the other complications.

Its importance lies in the fact that it is liable to occur in a mild case of Dysentery after the dysenteric symptoms have disappeared or, indeed, have been so slight that the patient may never have consulted a doctor. Thus there might be a risk of a carrier being able to spread infection, unless precautions be taken. French writers, who have drawn more special attention to this mild type, also point out an interesting syndrome of symptoms - Dysentery - Conjunctivitis - Arthritis. To the individual the importance of recognising the causal connection between Arthritis and Dysentery lies in the fact that it would lead to a definite investigation of the stools and recognition of the type of Dysentery, with the result that appropriate treatment would be adopted to prevent relapses and development of a chronic dysenteric condition. We have seen how a liver abscess as a complication was almost pathognomonic of Amoebic Dysentery, and some

authorities have regarded Arthritis as a complication in much the same light with regard to Bacillary Dysentery. It is, undoubtedly, perfectly correct to say that Arthritis is most frequently a complication of Bacillary Dysentery, but there are certain references to its occurrence in Amoebic Dysentery.

(a) Amoebic.

Though it is quite conceivable that the Entamoeba might be carried to the joints and cause a direct infection, still I have been unable to find any recorded instances of the Entamoeba being isolated from the joint. Such an authority as Osler says, an arthritis does arise sometimes in the course of Amoebic Dysentery, but that it is probably a toxic development. Certain other authorities have quoted instances of cases of Amoebic Arthritis, e.g. Moorhead quotes a series of six cases of Dysenteric Arthritis, in four of which there was positive proof of Amoebic Dysentery. In all the cases he found emetine rapidly curative. Dr. Madden likewise quotes another case of Arthritis complicating Amoebic Dysentery. In none of them, however, was a mixed infection with Bacillary Dysentery excluded. We have seen how this mixed infection of Amoebic and Bacillary Dysentery is not so uncommon. The fact of Emetine curing the Arthritis I don't regard as any conclusive evidence whatsoever, as my experience is that the arthritis disappears

with treatment on general principles, and that there is no specific treatment once it has developed.

The importance of the treatment with Emetine is in curing the Amoebic Dysentery and so preventing both the spread by a carrier and further complications such as Amoebic Abscess of the Liver.

So far as our present knowledge goes, there is no evidence that the *Entamoeba Histolytica* has any other action than a proteolytic one on the cells in its neighbourhood. There is nothing to support the idea of a toxin developed from it directly. In considering, however, the morbid anatomy of the intestine in Amoebic Dysentery we have seen how the necrosed foci are liable secondarily to an invasion by other bacteria, as is evidenced by the "septic" pyrexia sometimes observed in the later stages of amoebic dysentery.

In such cases certain of the numerous bacteria, normally saprophytic, may assume virulent activity, and enter the blood stream. Magner quotes three such cases out of a series of thirty blood cultures carried out in this later pyrexial stage. In one case *B. Coli Communes* was isolated on the 3rd day of pyrexia when *Entamoeba Histolytica* was isolated from an acute case of Dysentery. Another case of similar kind on the 3rd day yielded an unidentified bacillus - motile, Gram. negative, non-sporing. Does not liquify gelatin,

produced acid in glucose. No change in Lactose, Mannite, Dulcitol, Saccharose or Maltose. No indol. Agglutination:- feeble clumping with patient's serum 1-50 in 12 hours. No agglutination with Antidysenteric, typhoid or paratyphoid sera. Non-pathogenic to guinea pigs.

In an analysis of 300 cases of Dysentery he found that 8, or nearly 3%, were mixed infection with *B. Dysenteriae* and *Entamoeba*. As he points out, a further number of cases were probably originally infected with *E. Histolytica*, as many had been treated with Emetine before they came under his observation. Thus the percentage of mixed cases might even be higher. It has been noticed that cases of double infection are usually severe and it is in just such cases that toxic phenomena are common.

Accordingly, it is quite a reasonable conclusion to draw, that we may have in Amoebic Dysentery an Arthritis developing which is caused secondarily, as a result of either toxins developed by those bacteria, or by actual invasion of the joint through the blood stream.

(b) In Bacillary Dysentery it is an undoubted complication, and in my cases developed as a complication of both Flexner and Shiga Dysentery. In several of my cases the Dysentery was clinical, neither bacilli nor entamoeba having been isolated. An interesting

feature was the fact that a joint that had been previously damaged was more prone to suffer. Thus one of the cases had previously a synovitis from a displaced semilunar cartilage, and in two other cases there was a history of previous Rheumatic Fever. A French authority - Crouzon - in a recent series of 420 cases of Dysentery found 9 cases of Arthritis.

The only positive finding in the stools was a para-colon bacillus. Certain of his series, however, are open to the criticism, that they were serum joints as they occurred ten days after anti-dysenteric serum was given. Others were undoubted dysenteric joints, as they occurred where no serum had been given.

Case Incidence.

Varies very much with different epidemics. It was reported as being common among the British troops who suffered from Dysentery during the South African War. In a series of 757 cases reported by Bose in 1913 there were only 5 cases of joint trouble, whereas, according to Kelsch and Kiener, it occurs in 1-38 cases of Dysentery. Reviewing collected literature of a series of 2500 cases of Dysentery in recent years I find the case incidence to be 1-95. Rogers, on the other hand, states that he never had a case in his experience in India. The fact that Amoebic Dysentery is much more frequent in India probably explains this. My own series of 9 cases cannot count for per-

centage, as I cannot give the correct total of cases of Dysentery they were taken from. Where the case incidence is over 1% it is probable that certain cases of Serum Arthritis are included.

Predisposing Factors.

Previous injury to joint was present in one, and previous Rheumatic Fever in two others of my cases. In Clifford Allbutt's System it is stated, that previous Rheumatism or Heredity does not predispose to it.

Analyzing a series of 60 cases in Military Hospitals, 1918, Schittenheim notes that it frequently comes on after transportation when faeces were solid or at most only a little mucus.

In my own cases 6 developed an Arthritis while still in bed and under careful dietary. The other three were transferred from another Hospital while the joint condition was subsiding. In those cases the Arthritis developed after getting up.

Previous history of Dysentery was present in two cases. There was no Arthritis present during the first attack in either of the two.

Severity or otherwise of the Dysenteric Attack.

Many of the cases are described as occurring in the course of a very mild dysentery, in which dysenteric symptoms have cleared up. In one of my cases

the dysentery was a very severe one and the patient eventually died. One was a mild attack, while the other seven were of moderately severe type.

Serum treatment.

Does serum treatment predispose to joint affection or otherwise in Dysentery ?

Some authorities are inclined to regard most of these joints as serum joints. The fact, however, that in six of my cases no serum had been given, prior to the Arthritis, contraverts this.

There is a very characteristic type of Poly-arthritis that develops from 8-10th day after serum, but this is quite distinct from the true Dysenteric Arthritis.

Two cases which had serum treatment originally, both complained of fleeting joint pain associated with the serum reaction from 8th - 10th days, but the typical Arthritis did not develop till later. A certain proportion of serum treated cases complain of joint trouble of more or less severity during the period of serum reaction. As the two serum treated cases had distinct joint pains during the reaction period about the 10th day, it would seem as if there is a certain individual susceptibility to joint affection, and in those predisposed there is the risk of an Arthritis developing. During the summer and autumn period, 1918, when the cases were of a severe type

a very large number of my cases were treated with serum. A small number of those had serum joints, of whom 2 developed Arthritis, while two who had no previous serum likewise got an arthritis. During the winter period 1917-1918, the cases were of milder and more chronic type and I gave serum to a very small number. Of the four cases of joints during that period none had had previous serum treatment.

My own conclusion is that serum treatment does not provoke a true dysenteric arthritis, but, on the contrary, that efficient serum treatment helps to prevent its development. At the same time, if there is a distinct serum joint, it is advisable to keep a special lookout for a subsequent true Dys. Arthritis.

Pathology.

The characteristic of this condition is the presence of fluid in the joint to a greater or less extent. In five cases I aspirated the knee joint, and in four of them the fluid was absolutely sterile, no bacteria or parasites being discovered. In one there was a profuse streptococcal infection. The fluid is of a greenish straw-coloured synovial character, varying from clear to slightly turbid. In the streptococcal case it was very turbid.

The fluid usually contains polymorphonuclear cells. In studying the literature on the subject this seems to be the usual condition found. Suppura-

tion is unusual, except there be a mixed infection as in Case 7. Another such case is quoted by Bose, where there was a B. Coli infection of both knees and elbow.

Singer quotes a severe case where the joint fluid was sterile, although there was septicaemia - both staphylococci and streptococci being isolated on haemoculture.

Norf and Collard quote one case where the joint fluid agglutinated B. Flex. Y.

Associated with the fluid may be a certain amount of synovial thickening in some cases. In the polyarticular type there has been described an associated tenosynovitis in some cases.

In one of my cases which was a severe B. Shiga Dys. with joint infection, the joint condition subsided and became normal. This patient eventually died several weeks later. At the post-mortem the knee joint was opened and seemed perfectly normal.

In a previous paragraph the type of the bacilli has already been considered as primarily the cause of the dysentery. In the cases under consideration 4 were Shigas, 1 Flexner, 4 Clinical. Of the clinical one showed streptococcal infection. The findings in these cases, as in others recorded, seem to point to the action being due to a toxin, as the joint fluid is usually sterile. We know, moreover,

that the Dysentery Bacilli do produce a toxin which, in the case of B. Shiga especially, may be very potent and toxic. The usually accepted theory is, that the bacilli manufacture in the intestine/ ^{the toxin} which is absorbed and re-eliminated into the large intestine, where it has a selective action on the mucus membrane. During the third and fourth week the absorption of toxin may be modified by the assisted action of other saprophytic secondary invaders of the weakened mucus membrane. It is at this period of the disease that the selective action of the toxin or toxins pick out the joints and cause a certain amount of pyrexia.

CLINICAL TYPES.

Two types are met with,--

1. Polyarticular.
2. Mono or Diarticular.

The polyarticular type has been described by certain authors. Crouzon in a series of 9 cases describes the knees, tarsal joints and sometimes hip and shoulder as being affected. It occurs in his cases in 10-15 days from the onset of the Dysentery. The effusion persists a long time - several weeks. The type of dysentery was frequently a mild one. The condition occurred both with cases which had

serum and those which had not. Another feature of the cases he quotes was a combination with Conjunctivitis. This polyarticular type except as a serum reaction I have not seen.

Mono or Diarticular.

This is the more usual type and is the one that occurred in my cases.

It is almost always the large joints that are affected, usually the knee or knees. During the third or fourth week of the disease, when the bowel symptoms are clearing up and the temperature has been keeping normal, the first indication of trouble is a rise in temperature to about 100° and the complaint by the patient of pain in a knee joint. Fluid gradually accumulates in the joint, which, in the course of 48 hours, may be considerably distended. The pain in the joint, which is only moderately severe, depends on movement of the joint and on the amount of fluid. Great distension causes considerable pain. For the next four or five days there is a slight irregular pyrexia from 98° to 100° . There is little or no redness of the joint. After 48 hours the other knee joint, not infrequently, becomes affected likewise. In an acute case the joint may return to normal in the course of two or three weeks, the fluid gradually subsiding. A marked feature is

the great wasting of the muscle above the joint. In other cases there may be a certain amount of thickening of the synovial membrane with some stiffness and limitation of movement. Such cases are apt to be very tedious, but the ultimate recovery is usually good. In four acute cases which I was able to follow from the outset the joint recovery was complete in the course of a few weeks. A most important contributory factor to this result is, I consider, the treatment as if it were a surgical joint. This I shall refer to under treatment.

There is an associated condition, to which special attention has been drawn by French writers, which was present in two of my cases, namely,--
Conjunctivitis. This is sometimes a premonitory feature of the joint condition. Whenever Conjunctivitis is observed in a case of Dysentery during the third week it is a danger signal for an onset of Arthritis. It was Major Evans who first called my attention to this interesting phenomenon, and on two occasions this observation was confirmed. The Conjunctivitis first appears; and in the course usually of 48 hours the joint symptoms manifest themselves. The conjunctivitis then tends to subside and gradually disappears. In one of the cases (6) there was a slight purulent Blepharitis, and in the other case (8) an associated Iritis.

There was also Photophobia.

The conjunctivitis occurs without any history of Gonorrhoea. Rist, who has investigated such cases, states that the secretion does not contain Dysentery Bacilli or other organisms apart from those normally present in the conjunctival sac. He draws an analogy between this and the bilateral metastatic gonorrhoeal conjunctivitis which frequently coincides with an attack of Gonorrhoeal Rheumatism. In such cases the conjunctival secretion is sterile.

Crouson, likewise, from his recent experiences of the association of Dysentery with Conjunctivitis and Arthritis, concludes that, when the gonococcus can be eliminated as a factor, it is safe to diagnose Dysentery retrospectively in a case first seen with Conjunctivitis and Arthritis. As he points out, the original dysentery may have been mild or a chronic slight type. This associated condition may thus lead to the detection of a "carrier".

There was no indication of heart complication in any of my cases, nor has this been noted in recorded cases.

The extreme rarity of suppuration has already been commented on, but Case (7) shows how it may occur secondarily, owing to mixed infection. That there is this danger emphasizes the necessity for meticulous care in the aspiration of a joint aseptically. In

Case (No. 7) there was a pyaemic state consequent on the leg wound and the streptococci settled in a joint rendered susceptible by the Dysenteric Arthritis.

Accordingly, it is important to remember that in a dysenteric joint the fluid, though usually sterile, may be purulent from mixed infection, and require surgical treatment.

Serum Joint.

At this point I should like to draw attention to the clinical nature of this condition, which is apt to be confounded with a genuine Dysenteric Arthritis.

During this last summer I gave a large number of Anti-dysenteric sera, and a serum reaction, usually about the 7th or 8th day, was very common. In almost all cases there was more or less of an Urticaria with a mild pyrexia for two or three days. Sometimes there were fleeting joint pains, and in a small number of cases the joint condition was most acute. In such cases the temperature would rise sharply to 101° or 102° and very acute pain be felt in the small joints - it might be first the wrists, later the ankle and then spread to the large joints, - shoulder, elbow and knees. For 48 hours this polyarthrititis would be most acute, - the slightest movement giving great pain and the weight of the bed-

clothes causing extreme discomfort. There was no fluid in the joint. The condition seemed to be peri-arthritis in the fibrous tissues and ligamentous structures. In the knee, for example, a characteristic spot where the pain was first felt was in the popliteal space which was very tender to touch. In such cases slight flexion of the knee, by relaxing the fascia, gave considerable relief. This condition in the knee was in striking contrast to the true Dysenteric Arthritis, where the pain was not so great and the tenderness was more in the front of the joint over the distended capsule.

In the course of 48 hours the joint symptoms usually entirely disappeared. Sometimes they might persist a day or so longer and the pyrexia vary from 99° to 101° .

It was the rule, however, for the joint to return to normal in the course of a few days.

The whole condition then is in marked contrast to the clinical picture of Dysenteric Arthritis.

PROPHYLAXIS.

Having considered, up to this point, the effects produced on Man by the entrance into his system of certain parasites and bacteria and the contagious nature of the diseases they produce, one naturally asks,- What can be done to prevent the infection?

In all wars this has ever been a pressing problem. What prophylactic measures can effect is nowhere better exemplified than in the Russo-Japanese War, when the sick-rate was brought down to such a low figure. The Japanese attribute this entirely to the care taken by sending Sanitary experts ahead of the troops to investigate the conditions of Soil and Water Supply.

Water.

The importance of a pure Water Supply cannot be overestimated. We have seen how both bacilli and tetragenous cysts can survive in water, and how boiling quickly destroys them. The drinking of contaminated water is a very fertile source of infection. With troops in the field the modern Water cart with its filtration and chlorination, especially when combined with Horrock's test for estimating the measure of chloride of lime required for a particular supply, is a very efficient means of purifying water.

Unfortunately, if the particular water requires more than a small quantity of Lime, the water becomes very disagreeable to the taste. Much the most efficient means of purifying is by boiling. The habit of drinking tea in the army is a great safeguard for troops, as it should always ensure the use of boiled water. One point with regard to sterilization of water is important, namely, the need for care in protecting it after being sterilized previous to drinking. Vincent has shown that Bacilli Shiga survive longer in sterilized water than in contaminated water. Care is also required with water used for hygiene of the mouth just as with drinking. Cisterns, water-carts, and receptacles for storing water should be periodically cleaned with a strong solution of Chloride of Lime or Promanganate of Potash.

Pollution of Soil.

This is another frequent source of infection, especially, in camp life, and very strict disciplinary measures are required to prevent this. In Eastern countries the filthy habits of the natives in this respect is sufficient reason for the incidence of disease. Another pernicious custom is the cultivation of gardens etc. with house sewage. When this is done, especially, as in France where lettuce etc. are so much eaten, the spread of disease is

inevitable in the presence of the "carrier". It is beyond the scope of our present subject to more than mention those sources of pollution, as it introduces the whole question of sanitation and the proper disposal of excreta.

In dealing with those actually suffering from the disease the greatest care is required in disinfecting of bed-pans, commodes, bed clothes, etc. Cleansing in strong solution of cresol is efficient, but, when possible, it is advisable to use high pressure sterilization when it has to be done on a large scale, as in Hospitals. Similarly with bed-pans: a few minutes' boiling is desirable after preliminary soaking in cresol.

Flies.

Flies are the greatest curse, especially in hot climates. They swarm wherever food or faeces are about, and we have already seen how they can carry the bacilli and cysts. Accordingly, efficient protection of all food from flies and a good supply of fly traps are essential. In a word, when there are bad cases which are frequently using the bedpan, the greatest care is required to prevent flies becoming infected. The free use of cresol or formalin and the immediate removal of soiled bed-pans is important. Special precautions are required, likewise,

by attendants on the sick, such as disinfection of the hands in a solution of cresol after handling bed pans or soiled clothing. A similar precaution is also desirable after defaecation, especially among the inmates of a Dysentery Hospital.

Prophylactic Sero Vaccine.

This subject will be dealt with later.

Elimination of the "Carrier".

As a prophylactic measure this is one of the most important. All convalescents from Dysentery may be regarded as possible carriers. Accordingly, in our Hospital the custom was to send them to a Convalescent Camp for three weeks at least.

(1) In the case of Bacillary Dysentery.

The large majority are soon free of infection, but there is, as has been already seen, a small residue who continue to pass bacilli. Such cases are probably passing some mucus or mucopus in the stools at intervals, and, so long as that is the case, they should not be discharged. Quite apart from the danger to the community of such cases, there is the possibility of an exacerbation of the disease at any time in the individual.

Unfortunately there is no specific treatment for the eradication of the "bacilli", such as there is in the case of the Amoebic carrier.

(2) The Amoebic "Carrier".

The prevalence of the "carrier" may be gathered from the following figures:-

Among 1979 healthy British troops in Alexandria, whose stools were examined by Wenyon in 1916, 106 or 5.3% were found to be carriers of cysts of *Entamoeba Histolytica*. Of these only 16 had suffered from slight attacks of Diarrhoea with abdominal pain. They were, of course, living in a country where Amoebic Dysentery is prevalent and consequently liable to infection.

A further series of 785 persons, who had never been out of Great Britain, were examined once. Of these 281 were patients in the Liverpool Royal Infirmary, and among them two positives were found, i.e. .7%. Of the 504 who were young recruits in training in a camp near Liverpool, where were also some men who had returned from the Mediterranean area, there were 24 positive with only one examination, i.e. 4.8%. The presumption is that many of these men got infected in the camp.

From a large series of cases convalescent from Dysentery from the Mediterranean in 1915 the Medical Research Committee publish the following figures:-

Entamoeba Histolytica in 12.6% as a result of three examinations. It was further found that the figure increases in a definite ratio, according to the

number of examinations, and that six examinations are necessary to get a correct idea of the incidence.

This would make the incidence 18-25%.

These were cases who had in many cases been treated with emetine and who had had the disease for some time. The specific treatment will be considered later.

The general precaution, however, in all cases of Dysentery, whether Bacillary or Amoebic, should be taken, namely, that none should be engaged in the handling of foodstuff or in cooking for a prolonged period after the attack, say a year. This is of great importance in the army. The small residue of persistent "carriers" should never be allowed to act as cooks, or servants handling food etc.

So much for general prophylactic measures.

Personal Prophylaxis.

In this respect much can be done to prevent infection, even though one is living in an infected area. Those measures are merely what commonsense would dictate; such as keeping the digestive system healthy by eating good and well cooked food, avoiding alike excess and starvation, avoiding uncooked fruit or vegetables, being moderate in the use of alcohol, by a regular daily evacuation of the bowels, by precautions likewise against over-fatigue, changing clothes after being wet,

in the Tropics the use of an abdominal belt.

The fact that so many of these ideals are impossible of attainment during active service conditions accounts, in part, for the prevalence of Dysentery in War time.

TREATMENT.

General.

During the acute and sub-acute stages of Dysentery, the patient should be kept warm in bed and not be allowed up for any purpose whatsoever. The bedpan and urine bottle should be warmed before use.

The patient should not be allowed up till the stools are approaching normal. Even then the greatest care is necessary, in the way of warmth etc., to prevent relapses, which are very apt to occur with the least indiscretion.

Diet.

Suitable modification of Diet is the fundamental principle in the successful treatment of Dysentery. In order to do this satisfactorily, it is necessary to examine the stools daily and modify the diet accordingly.

In the acute stage plenty of bland fluid drinks is desirable, preferably in small quantities at a time at frequent intervals. The drinks are better when warm, as too hot or cold drinks are apt to excite colic.

Fluids such as barley water, albumen water, rice water, chicken tea, beef tea, tea, are suitable for the first few days. Milk seems to be better avoided in the early stage. When it is given later, it is advisable to dilute it with barley water, and adding Sodium Citrate gr. ii to the ounce. Raw eggs, beaten up and taken in tea or beef tea, are very useful after the first few days.

Where this acute stage is prolonged it is advisable to give sugar freely in the drinks. Glucose solution is valuable.

Various forms of jellies, e.g. plain, milk, or chicken may be added after the first few days. Later, when the acute stage is subsiding, Benger's Food, Horlick's Malted Milk, custards, arrowroot may be added.

Well boiled rice added to the beef tea is a welcome addition. Similarly a few plain biscuits very carefully masticated or dry bread crumbled up in the beef tea are grateful. When one considers that the injured part is in the large intestine or only a very small lower part of the upper, there is not the same care needed in avoiding a little bread or biscuit as there is in Typhoid. The important thing to avoid is bulk of residue.

When the stool is free of blood and perhaps there is only a little mucus with a soft or semi-formed

stool, chicken form may be added, and later minced chicken or minced meat with rice. Bread and butter is gradually increased. The greatest care is required in allowing vegetables and an ordinary diet. These additions should be very carefully checked by observing the stools. One frequently found undigested particles of food being passed, necessitating further care. It is very important to emphasize the importance of proper mastication. Unfortunately some of our patients had very defective teeth which rendered dieting difficult. The routine in Hospital was that the man had to be able to take an ordinary diet for eight days, during which time his stools had to be normal, before he could be considered fit to be discharged to a convalescent camp where he was kept for a minimum of three weeks before being discharged to duty.

In some districts "Yaghurt" or sour milk has been used very successfully in dietetic treatment. Among the Serbs, I understand, this was used in the Hospitals. In Egypt Drs. Willmore and Savage have also used it with good results. The following is a description of how they prepared it scientifically:- Several samples of sour milk were inoculated with sterilized milk. That which gave the softest and most homogeneous curd was then plated on McKonkey or Lactose, cochineal agar. A pure culture was

obtained of a large lactic acid producing bacillus and a living streptococcus. These were inoculated into sterilized milk, and each batch of 'Yaghurt', prepared in covered sterilized jars or incubators at 37°, was inoculated with a flamed spoon from the preceding.

Seeing that lactic acid is antagonistic to the growth of Dysentery bacilli, this would seem to be a very suitable and satisfactory form of nourishment.

The acute case usually does well on a diet as previously described. If, in the course of the illness, a complication should arise, such as perforation or haemorrhage, it will be necessary to stop food altogether by the mouth, except for sips of albumen water or plain boiled water. In the case of the former there will probably be surgical intervention and consequent low dietary. In the case of haemorrhage, in the course of 24 or 48 hours it may be possible to begin and gradually increase the dietary along the lines of that for an acute case.

If there is a scorbutic element in the acute case it is well to give orange, or lemon, or lime juice, or grape juice. The milk that was available in Salonica was all tinned, so there was no antiscorbutic principle in it.

The general treatment for the Chronic Convalescent case is on the following lines: He may be allowed

out of bed and take gentle exercise, but should be specially protected against chills.

The diet should be easily assimilable and non-irritant, consisting chiefly of soups, pudding, eggs, minced chicken or minced meat with rice. Bread and butter should be allowed sparingly. Milk in the form of Benger's Food or Horlick's malted milk or even plain citrated milk. The most valuable item in the diet are eggs, and, if two or three can be taken per diem without upsetting the digestion, so much the better.

Medicinal.

In the routine treatment of cases of Dysentery occurring in Salonica one had to consider in every case, what malarial element there was in the case. If the case were an acute one with pyrexia, it was advisable to take a blood film, and, if malaria parasites were present, quinine was necessary. In the acute stage of the Dysentery the intra-muscular method was desirable, as quinine by the mouth aggravated an acute dysentery. Moreover, with the frequent action of the bowels, it was apt to be passed through the intestinal canal too quickly for absorption. Throughout the further course of the case, once the dosage of quinine had been gradually reduced, it was better to keep up a small dose of Quinine Sulphate by the mouth.

Further diagnostic help might be got from the blood film towards the diagnosis of the type of Dysentery, by noting the presence or absence of leucocytosis. When this was absent the case was, in all probability, Bacillary Dysentery; whereas, if there was a distinct leucocytosis 15,000-20,000, the case was in all probability Amoebic.

A specimen of the first stool passed is sent for examination at a laboratory, for diagnosis as to whether it is Bacillary or Amoebic.

Having considered the case carefully as regards acuteness of onset, pyrexia, previous history of Dysentery, type of Dysentery prevalent in the locality, character of the stool etc., it may be possible to make a shrewd guess as to the type. The further treatment will depend on which it is proved to be by the laboratory finding. The possibility of it being a mixed infection must be borne in mind. The general treatment common to both types of Dysentery has already been considered.

(1) Medicinal Treatment of Amoebic.

A preliminary dose of Castor Oil $\frac{3}{4}$ i with xxm Tinct. Opii or Dover's-powder *gr V* at the very outset. The Saline treatment, which is almost specific for Bacillary Dysentery, is also useful in Amoebic cases, as it helps to keep a flow through, and free irrigation of, the intestine, sweeping away parasites. Sodium

Sulphate $3\dot{i}$ two hourly in the acute stage, and after 24 or 48 hours reduce it to $3\dot{i}$ four hourly, and later $3\dot{i}$ T.I.D. or as required. The important principle is to keep a regular soft action of the bowels once or twice a day, so as to prevent irritation of the ulcerated mucous membrane.

The specific treatment for Amoebic Dysentery spells Ipecacuanha or its derivatives. Formerly, before the discovery of the value of Emetina, the Ipecacuanha treatment was very cumbrous and nauseous. The following is the method of treatment with Ipecacuanha as described by Manson,-

"Interdict all food for three hours. Then give 10 - 20 drops Laudanum in a tablespoonful of water, and at the same time apply mustard poultice to epigastrium. About 20 minutes later give 20 - 30 or even 60 gr. Ipecacuanha in pill, bolus, capsule or in suspension in about half a wine-glassful of water. "

To prevent vomiting, the patient is directed to lie flat on his back, using a low pillow, and not to eat, drink, speak or move for at least four hours. After six or eight hours small quantities of food may be given. Continue Ipecacuanha treatment nightly for a week reducing the dose daily by 5 grains. Thanks to the investigations of Vedder in 1910, the Amoebicidal properties of Emetine, the alkaloid of Ipecacuanha, were proved. Rogers confirmed its therapeutic value by its use in the treatment of Amoebic Dysentery in India. With the further experience of the last few

years, its efficiency in one form or another has been abundantly proved.

In further considering the methods of treatment adopted, it is necessary to remember that the objects to be achieved are two-fold:-

1. To alleviate the acute symptoms and cause healing of the damaged intestine.
2. To eliminate the parasitic infection; or, in other words, free the carrier of cysts.

In order to achieve the first of those results, Emetine hydrochloride given hypodermically in 1 gr. dose for 12 days has a wonderful effect. Wenyon and O'Connor recommend 1 gr. Emetine hypodermically supplemented by $\frac{1}{2}$ gr. by mouth daily for 12 days.

It has been found, on investigating a large series of cases treated with Emetine, that 70% of the cases remain positive carriers of cysts. Accordingly, even though clinically the cases seem to have been cured by this specific drug, the infection is not necessarily eradicated. Where insufficiently treated there is not only the danger to the individual of relapse and further complication, but also the risk of spreading the disease to others.

This brings us to the further methods to be adopted to achieve the second result. The following embodies the most approved and authoritative statements published by the Medical Research Committee:--

Attention is called to the necessity of examining the stools at definite periods. Six negative examinations must be got by an expert protozoologist after the first course of treatment is completed.

There must be a negative obtained at least 21 days after completion of the course. It is recommended that during the 1st week there should be 1 negative;

,,	2nd week	,,	1	,,
,,	3rd week	,,	4	,, .

It has been found that the majority of the cases which relapse, do so before the end of the third week.

As soon as a relapse is found, a second longer course of treatment is adopted.

The method of administration of the drug recommended is the insoluble double Iodide of Emetine and Bismuth by the mouth, - 3 grains per diem for twelve consecutive days. Acute and Subacute cases are treated in bed, but "carriers" don't require to be in bed. Administration on an empty stomach is not advisable. The patient should go to bed after a good meal, and the dose should be taken with a hot cup of tea. If there be vomiting, Tinct. Opii m. X - XV should be given shortly before the Emetine. The best results were got by giving the drug as a loose powder in a small gelatine capsule. With this method, it was found that 90% carriers were cured of the infection.

When this first course failed, a further more prolonged second course of 3 grains per diem for 24 consecutive days is recommended. With this further treatment there still persists a small residue of infected "carriers". The conclusion is, that this residue does not constitute more than 5% of all carriers. It has to be noted that these investigations are made on a series of difficult resistant cases which had, in most instances, been treated originally with one or more courses of Emetine. If one takes a percentage of the actual carrier from all the cases of Amoebic Dysentery, it would be very much smaller.

The good results attained by the double iodide in the treatment of "carriers" has made authorities consider the application of the double Emetine Bism. Iodide for acute cases. In the small number of cases where it has been used it seems to have been efficacious they state.

Other forms of Emetine have been tried, but results are not so successful as with the above: e.g., Methyl Emetine and "Alcestra" which is a combination of Ipecacuanha with Fuller's Earth.

Such then is the specific treatment for Amoebic Dysentery. Occasionally Dover's Powder 5 - 10 gr. may be useful or even Morph. $\frac{1}{4}$ gr. hypodermically when the stools are very frequent and colic is severe.

In such cases heat to the abdomen, in the shape of fomentation or turpentine stupe, is comforting. When tenesmus is distressing, a starch & opium enema is very satisfactory, e.g.- m xx Tinct. Opii in Starch with which may be combined 3ii Bismuth. In the early stages bowel lavage is useful in very acute cases: E.g.- Sod. Bicarb. 3i in a pint of warm sterile water succeeded by a weak Permanganate solution.

In the Chronic type, when there is ulceration which is slow to heal, various forms of enemata have been recommended, such as Silver Nitrate $\frac{1}{2}$ -1 gr. to ounce distilled water, Eusol solution, Sulphate of Copper, etc. The greatest therapeutic agents, however, are the specific drug combined with careful hygienic and dietetic treatment.

Treatment of Complications of Amoebic Dysentery.

(1) Amoebic Abscess of the Liver.

We have already noted that an abscess rarely develops when the case has been efficiently treated with Emetine in the pre-suppurative stage of Hepatitis. In the cases, however, which do develop into an abscess, further treatment is required. The procedure which has been adopted so successfully by Rogers is the following:-

The site of the abscess having been localised, and the skin at the site of puncture having been carefully sterilized, a full-sized aspirating needle is inserted, and as much as possible of the thick pus withdrawn. Some of the first pus to come away is collected in a sterile tube for examination. Then a solution of Emetine 1 gr. to $\frac{3}{4}$ or $\frac{3}{16}$ sterile water is injected into the abscess cavity. The cannula is withdrawn and a collodion dressing applied. After this simple operation some cases have been cured. In other cases it may be necessary to repeat the operation in a week's time. Occasionally even three or four times it may have to be repeated, in the case of very large abscesses.

The pus is examined each time. In his experiences it is sterile in about 85% cases. When sterile originally, it almost invariably remains so.

Originally Rogers recommended a solution of Bihydrochloride of Quinine of a strength 10 gr. in 1 ounce of water. Where large abscesses were emptied, he has used as much as 40 gr. quinine.

The principles underlying this method of treatment are:-

1. That in a large majority of the cases of Tropical abscess of the liver Entamoeba Histolytica is found in the wall of the abscess cavity.
2. That the abscess is usually sterile of other bacteria.
3. That /

3. That Solution of Quinine 1-500 readily destroys amoebae in the wall of such an abscess in vitro. (Rogers noted this in 1902.)

Similarly a very weak solution of Emetine is very powerfully amoebicidal.

In cases where the abscess proves not to be sterile, it must be laid open and drained on surgical lines. The method will depend on the site. The large majority are single abscesses, and most frequently in the right lobe. In these cases the best access is got by the transpleural thoracic route. McDill reports 90% recoveries from this operation when the abscess is single. He prefers to expose the liver and postpones a further stage of the operation for a day or so till adhesions form.

With improved surgical technique and asepsis there is not the same danger in this operation, as there once was, from mixed infection.

On account of the risk of haemorrhage occurring ~~xxxxxxxx~~ in a congested liver during the exploration commonly adopted to locate the abscess, some surgeons prefer to expose the liver first, as above described, or by abdominal route, and then explore the liver openly. It is important to continue treatment with Emetine after the operation.

I have not seen an abscess of the liver and so have no experience of this complication.

(2) Intestinal Haemorrhage.

Intestinal Haemorrhage has been mentioned as a complication of Amoebic Dysentery. In the case (11) already quoted, I found the most wonderful effect from Emetine hypodermically as soon as the parasite was recognised. Combined with this the case should be treated on general principles,- Morphia $\frac{1}{4}$ grain hypodermically and repeated if necessary. Nothing by the mouth except sips of boiled water or albumen water, and Calcium Chloride, 10 gr., was given four hourly. Adrenalin solution per rectum.

(3) Perforation.

Where this occurs in an acute gangrenous case, operative interference is hopeless. In the more chronic case with isolated ulcers operative procedure is more hopeful. There must however be no delay.

Treatment of Bacillary Dysentery.

What has been previously said as regards Quinine treatment where necessary, a dietetic and general treatment, likewise applies to Bacillary Dysentery. We now have to consider the further medicinal and special treatment to fight the toxin of the disease.

The Saline treatment, as usually employed, gives most excellent results and, combined with suitable dieting and rest in bed, is quite sufficient to cure a number of mild cases and even severe cases also.

Sod. Sulphate $3\bar{i}$
 Acid Sulph. Dil. $m\bar{xv}$
 Tinct. zingiberis $m\bar{v}$
 Aq. menth. pip. *ad 3ss.*

is a useful prescription repeated two hourly till the stools begin to get more fluid. In 24-48 hours the the interval may be increased to four hourly and then three times a day. In order to keep a soft stool which would not irritate the ulcerated mucous membrane, I found many cases required this dosage three times a day for several weeks. In some cases it could be reduced to once daily. As constipation is a most troublesome feature of convalescence in Dysentery, a mild saline aperient was required for some time.

Sod. Sulphate seems to be the more desirable salt, as it is milder in its action and has more of an action on the liver. Mag. Sulph. however does very well when the other is unavailable. A combination of the two answers well during convalescence if a rather stronger action is required.

What has been said previously of Sedatives and of lavage applies equally in Bacillary Dysentery.

Serum Treatment.

In an acute moderately severe case the question of the advisability of serum treatment arises.

During the summer and autumn 1918 it was my practice to give serum in all acute cases which were moderate or severe unless contra-indicated. It, undoubtedly, relieves the symptoms and the patient is more comfortable in the early stages. Anything which contributes to this and gives him rest from the distressing tormina and tenesmus helps to conserve his energy and accelerate convalescence. My impression certainly was that, if Serum treatment could be commenced early in an acute case, it made a more rapid and satisfactory recovery.

To attempt to give comparative figures from my own experience of serum treatment would be very misleading. For example, during the Winter 1917-18 as the cases were of a milder and more chronic type, I seldom gave Serum; whereas, in the following summer, the cases being of a much severer type, serum treatment was frequently adopted. During this period, likewise, there were the various complications of Malaria and Influenza. The mortality was distinctly higher in the latter series, but the concomitant disease contributed largely to that. So, to compare the results of those cases treated with serum with those not so treated would be utterly misleading.

The serum originally employed by Shiga was obtained by immunization of horses with the soluble toxin or bodies of the Shiga Bacilli themselves. Later the value of Polyvalent serum was recognised. This was made from Flexner Bacilli and various subtypes. It is now recognised that the value of the Polyvalent serum is enhanced when it is prepared from various strains of Bacilli prevalent in a particular epidemic in one locality.

The serum is usually administered subcutaneously or intramuscularly. It may also be given intravenously in severe dehydrated cases while saline is being given at the same time. The site of injection should not be the abdomen as is frequently done. If given here the subsequent tenderness interferes with examination of the case each day. It is preferable to give it in the lumbar region, or axillary region. Some prefer to give it intramuscularly in the thighs.

Dose. The dose I usually gave was 40 cc.- 60 cc. daily for three days. Less than 40 cc. seemed to be of little use. Some others preferred to give large dose 100 cc. once and not repeated as a rule.

As much as 100 cc. on six successive days have been reported without any untoward result.

It is necessary to make special inquiry as to whether the patient has had serum previously or is

subject to Asthma. With such patients the reaction is apt to be severe, and there is danger of serious Amphyllactic phenomena.

Many of our patients had had Antitetanic serum previously. Provided special precautions are taken, the danger can be largely minimised. Thus it is advisable to inject 1/100 grain Atropina as a preliminary. The serum should be given in 1 cc. first and followed in $\frac{1}{4}$ hour by 5 cc. and gradually give the full dose.

I have not seen any death from Anaphylaxis nor have I seen any trouble when the above precautions have been taken in suspected subjects. One case I can recall which suffered from Anaphylaxis and very nearly died. He gave no history of having ever had any serum given before. On subsequent inquiry I discovered that he could recall an illness of his throat three years previously for which he had something injected. So I presumed it was Anti-diphtheritic serum. Another case showed a curious late reaction which further illustrates the certain amount of risk there is in Serum treatment.- It was a moderate case of Shiga Dysentery which had 40 cc. A.D.S. for three days. About the 16th day he had the usual serum rash, but, being in bed, showed no other effect. He had the usual Calcium Lactate 10 gr. four hourly, and in the course of a few days was all

right. He was quite convalescent and stools normal in three weeks from the onset, when the serum rash reappeared and he had a nasty syncopal attack, being almost pulseless when seen. Fortunately he got over this and made a good recovery.

The treatment of the ordinary Serum reaction consisted in the administration of Calcium Lactate gr. 10 two hourly or four hourly as required on the first appearance of a rash, Locally a calamine lotion or Sod. Bicarb. paste was soothing, The irritation was intense at times. The serum joints were very painful and unrelieved, or at least very slightly, by Salicylates. Local anodyne application and protection from the weight of the bedclothes gave most relief.

As regards the period of giving Serum, the most efficacious is at the commencement. After the first few days, if the stool were still acute, I gave it; but, in the later stages, it does not seem to be indicated unless in a relapse.

In considering the result of Serum treatment, it is necessary to remember that only a certain percentage of cases are proved Shiga or Flexner. A certain proportion remain Clinical. It is in such cases that the particular strain of Bacillus that is causing the disease may not be included in the Polyvalent serum, with the result that the effects may be

disappointing. Furthermore, cases which do not seem to improve as they should, may prove to be mixed infection with *Entamoeba Hist.* Further examination of the stool should be made, even though previous examination has revealed a positive Bacillary and negative Amoebic result. Failing a positive finding, it may be desirable to try a short course of Emetine empirically. The Emetine is valueless for Bacillary Dysentery, but there is the possibility of an Amoebic infection which microscopic examination has failed to reveal.

The use of Human Serum has been favourably reported on from Germany recently. The following is the statement of procedure:-

A patient is taken who has passed through a very severe attack of Dysentery the same as that affecting the recipient.

100 c.c. blood is withdrawn from vein of donor.

30-40 c.c. of separated serum is injected the following day.

The results sound excellent and the principle seems a good one, but the withdrawal of 100 c.c. blood from a patient who has just recovered from a serious illness is hardly to be recommended.

Vaccine Therapy.

By means of the anti-toxin a passive immunity has been produced, and various efforts have been made

to produce an active immunity by means of a vaccine.

The great success of T.A.B. vaccine stimulated authorities to try a sero vaccine prophylactically against Dysentery. Accordingly a memorandum was sent out by the War Office in 1917 on prophylactic inoculation against Bacillary Dysentery. It states that the vaccine consists of an emulsion of several strains of B. Dys. Shiga, Flexner and Y., and is given mixed with a specially prepared serum which neutralises the toxin of the B. Shiga without interfering with the power of the vaccine to produce protective substances in the blood of inoculated individuals.

Two doses were given at an interval of 7 days. The first dose consists of .25 c.c.m of vaccine and .25 c.c.m serum. These two are mixed in the syringe and injected subcutaneously. The second dose is double the first. A third dose is advantageous.

Results are:- Reaction produced is slight. Agglutinins are produced in the blood which have persisted for some months. The agglutination titre of the serum of inoculated men is higher than that seen after an attack of Dysentery.

H. Graeme Gibson in 1918 reports on the results of the use of this in the field.

1500 men were inoculated and compared with 1500 not inoculated and under similar conditions.

Observations, which were made over a period of

16 weeks, prove that the incidence of Dysentery was markedly less amongst inoculated men than amongst uninoculated - 9 cases as opposed to 66.

The deduction drawn is, that wholesale prophylactic inoculation is undesirable, but that it might be a wise measure at the beginning of the summer in a district where Bacillary Dysentery is common.

Therapeutic use of a vaccine is reported on favourably by some authorities, especially German who have used a compound vaccine called "Dysbacta" .. Boehneke.

10 cc. serum is injected 1st day,

.3 cc. vaccine ,, 2nd day, and

increased by .2 cc. daily till stools become semi-solid.

In Chronic cases they commence with -

20 cc. serum

.3 cc. vaccine next day,

.5 cc. ,, 3rd day

.5 cc. ,, 4th day.

Good results are claimed from its use in acute and chronic cases.

In October 1918 further reports from German sources are to hand regarding this vaccine. The conclusion seems to be, that, when there are severe local phenomena associated with General Toxaemia, results are encouraging from 1 dose of serum followed by 3 or more doses of vaccine.

Where local phenomena predominate and general disturbances are slight, serum is unnecessary, while

vaccine has a very favourable effect. Owing to its immunizing effect the organic resistance is raised and convalescence shortened.

Personally I have had no experience of vaccine therapy in Dysentery. The adverse criticism I should make regarding the prophylactic use of a sero vaccine is that, in the event of the patient getting Dysentery and requiring to have a serum, there is the slightly increased risk of anaphylaxis after the serum.

Other therapeutic measures.

In cases, where there has been considerable dehydration and the sp. gravity of the blood thereby raised, considerable benefit is got from Saline infusion 1-2 pints subcutaneously daily for several days. Sometimes Intravenous saline in extreme cases is beneficial, but the subcutaneous infusion seems to be more satisfactory as a rule, as the fluid is rapidly lost again in intravenous infusion. As an emergency measure, however, it may be life saving.

In the Acute Choleraic form Rogers advocates 3-4 pints Hypertonic Saline intravenously. Recent researches emphasize the value of Gum Arabic solution in Saline given intravenously. By this means the osmotic pressure is maintained, so that the fluid does not leave the vessel so readily.

Enemata.

In the acute case the use of a Sod. Bicarb. solution lavage followed by a Permanganate solution has been mentioned. Similarly the sedative use of a lead and opium enema.

Similarly in the chronic case more astringent lavage may be tried, such as Silver Nitrate $\frac{1}{2}$ -1 gr, to $\frac{3}{4}$ i.^{ss} Protargol, Copper Sulphate 1-1000, Tannin, Alum, Eusol, etc.

Personally I have not found very great benefit in the cases where they have been used, except in cases where there is evident ulceration in the rectum with a tendency to haemorrhage. In such cases Copper Sulphate in Alum sol. and Adrenalin are indicated.

Appendicostomy.

Appendicostomy is a treatment that has been advocated by some authorities and appears to have been successful in several cases.

In view of the fact, however, that the condition of the walls of the gut in Dysentery are in poor condition and unfitted for operative procedure unless unavoidable, it seems inadvisable to attempt an operation such as appendicostomy for this condition.

With /

With regard to the use of tonics etc. during convalescence from Dysentery, there is a great difficulty in administering tonic drugs. Being so frequently combined with Malaria with a resulting anaemic state one felt that Iron in some form was desirable. My experience was that Dysenteric symptoms were liable to be provoked by the usual Iron and Arsenic mixtures.

Alcohol.

During the acute stages it might be necessary to give Brandy, but, generally speaking, it was better avoided. During convalescence Stout seemed to be beneficial in the chronic debilitated cases, especially if anaemic from malaria as well.

Treatment of Complications of Bacillary Dysentery.Arthritis.

The most important consideration is in securing absolute rest for the joint.

If there be fluid in the joint, e.g. the knee joint, it is advisable to aspirate the joint. At first I only did this as a palliative measure to relieve the distension of the joint. In this way not only is pain considerably relieved, but the ligamentous structure and capsule of the joint do not remain over-distended. Consequently the lax. joint later is avoided. Later experience emphasized the advisability of aspirating every joint with fluid, as in Case (7). In that case the joint at first was treated by rest, etc. It was only when the joint seemed to be over-distended that I aspirated and found a profuse streptococcal growth.

The aspiration must be done under the most rigid aseptic precautions. The skin is carefully disinfected with Tinct. Iodine. A long sharp needle attached to a 20.cc exploring syringe is inserted into the joint above the patella, and as much as 3-4 ounces fluid may be withdrawn. Some of this fluid is injected into a sterilized tube and sent to the laboratory for examination.

The puncture is sealed with collodion; sterilized dressing and a quantity of wool applied and firmly

bandaged with a flannel bandage. This pressure with wool and bandage is important, as it aims at preventing re-accumulation of fluid. The limb is then put up in a semiflexed position on a M'Intyre splint.

The fixation of the limb gives great relief and further, it aids greatly in mitigating the joint trouble.

In 24 hours the fluid may reaccumulate and, if distension is marked, may require to be tapped again. As a rule the reaccumulation is not marked.

Painting the joint with Liniment Iod. daily is beneficial as a counter-irritant. After a few days passive movement of the joint may be commenced very gradually combined with massage of the muscles above and below the joint.

Gradually, as the fluid subsides, the joint returns to normal. Daily movement and massage are continued.

For the first 24-48 hours the joint may be painful, and this can be soothed somewhat by lead and opium application. As a rule, however, the relief from the aspiration of fluid is considerable and no further anodyne required.

Drugs such as Aspirin or Salicylate don't seem to affect the joint condition.

Serum.

I employed Serum in one case (5) when the Arthritis had developed. The joint condition did very well, but I have seen other cases do equally well without serum. The reason I employed serum in this particular case was because originally the case had not had serum treatment, progress was unsatisfactory and the joint trouble developed. Anti-Dysenteric serum, 40 cc., was given for three days.

The general opinion is that Serum is of no use in Arthritis, inasmuch as this complication occurs in the later stages of the disease. When serum is given however in the early stages of the Dysentery, not only is the chance of Arthritis developing diminished, but, if an Arthritis does supervene, it appears to run a milder course. Where there is mixed infection, such as Streptococcal, the joint requires to be opened and drained on surgical lines.

Those cases of Arthritis which I have seen arising in the course of an acute attack, while the patient has been in bed, have done excellently well. The condition subsided and the joint became normal again. In some cases, however, there may be some more persistent thickening and stiffness requiring perseverance with massage and movements.

By early and careful attention to a Dysenteric joint these untoward results are averted.

Intestinal Haemorrhage.

The same general measures are necessary as indicated under complications of Amoebic Dysentery. It has been claimed for Serum treatment that the horse serum, by increasing the coagulative power of the blood, diminishes the risk of haemorrhage.

Gelatin $\frac{1}{2}$ dr. dose four hourly, I found useful on one occasion combined with Calc. Chloride 10 gr. four hourly.

Adrenalin injections by the bowel are of use when the haemorrhage is low down.

Morphine hypodermically is, however, the sheet anchor for haemorrhage, as it keeps the bowels at rest. At the same time one must guard it against producing constipation with a resulting further irritation of the ulcerated area. So it is well to keep up a fluid exudation into the intestinal tract with mild doses of Sod. Sulphate carefully given.

In the event of much loss of blood, it may be necessary to give Saline infusion subcutaneously.

Ergotin may be given hypodermically and also Pituitrin 1 cc, and Strych. hypodermically is useful, if there is any collapse.

Parotitis.

When suppuration is present, the abscess must be opened freely and well drained on strict antiseptic

lines. In these cases the local condition clears up rapidly with this treatment, but the general condition is usually bad. There seems a tendency for this complication to arise as a terminal phenomenon. Where it arises in Amoebic Dysentery the specific Emetine treatment is important.

SUMMARY and CONCLUSIONS.

1. In addition to the types of Dysentery caused by the Protozoa and Bacilli respectively, it is necessary to recognise a Clinical Dysentery in which the actual organism has not been isolated.
2. The chief protozoon is the *Entamoeba Histolytica* which has a definite life cycle. It is recognised by its morphology under the microscope. It is non culturable. The most prolific cause of the spread of the disease is the 'cystic' stage of this parasite which is passed in the faeces.
3. The distinctive pathological feature of Amoebic Dysentery is the invasion of the submucous coat of the large intestine by the active *Entamoeba Histolytica*. This leads to necrosis of the mucus membrane and ulceration, which may penetrate into the deeper coats and even cause perforation. Further complications are caused by the parasite entering the minute branches of mesenteric veins, and thence, being carried to the liver, may lead to Hepatitis, and, sometimes, Abscess of the Liver.
4. The Bacilli Dysenteriae are recognised by their Morphology, Culture, especially their sugar reaction, and agglutination test. The two main groups are Shiga and Flexner with numerous sub-types and strains.

5. Bacilli are found in the stools. They disappear fairly early as a rule in an acute case, but sometimes persist for six months. They have been found in the blood in some cases.
6. The agglutination test is an important aid to diagnosis and identification of a special bacillus. It cannot be relied upon by itself, but must be viewed in conjunction with other tests.

It is important to standardize both methods and strains of Bacilli with controls. The agglutinins are not present during the first week and may persist for six months.

7. The characteristic feature of Bacillary Dysentery is the elaboration of toxins by the bacilli which have a selective action on the mucous membrane of the large intestine and, in some cases, lower part of Ileum. As a result inflammatory and ulcerative changes occur in the inner lining of the bowel. Ulceration has a greater tendency to remain superficial in Bacillary Dysentery.

Further complications are caused by the action of the Toxins.

8. In all types of Dysentery the Clinical picture is modified and complications caused by the secondary invasion of the diseased mucous membrane by other bacteria which may be normally present in

the bowel.

9. There may also be a mixed infection of the Amoebic and Bacillary type.
10. In Malarial districts the clinical picture is frequently complicated by the presence of a malaria attack.
11. In both Amoebic and Bacillary Dysentery acute and chronic forms are seen. Chronicity is more a characteristic of Amoebic, and acuteness of Bacillary Dysentery.
12. Sometimes the Dysenteric symptoms are so slight or 'latent' that the diagnosis may not be made till a complication ensues, e.g. Amoebic Abscess of the Liver or Dysenteric Arthritis.
13. Arthritis is a definite complication of Bacillary Dysentery. Its occurrence in pure Amoebic Dysentery has not been proved.
14. Evidence seems to be in favour of its being the result of the action of a toxin. Bac. Dysenteriae have not been found in the joint fluid which is usually sterile. Sometimes suppuration occurs, but this is due to mixed infection.
15. Arthritis occurring in the 3rd or 4th week of the disease is to be carefully distinguished from

serum joints occurring from the 8th-10th day.

16. Sometimes a Conjunctivitis is associated with a Dysenteric Arthritis, preceding it as a rule by a day or so. With the onset of the Arthritis the conjunctivitis tends to subside, but may reappear after the joint symptoms have gone.

17. Prophylaxis is most important in both types of disease. Especial care is required in protecting water and soil from contamination.

The elimination of the 'carrier' in both Bacillary and Amoebic Dysentery is most important. Recognised carriers should be ear-marked and should not be allowed to handle or cook food.

18. There is no specific treatment for eliminating the Bacillary carrier, but there is for the Amoebic who should be subjected to a thorough course of treatment with Bismuth Emetine Iodide. It is recommended that he be not declared free of infection till he has had six negative examinations made of the stools for at least three weeks after treatment.

19. Diet is a most important element in treatment. The careful observation of the stools is the only sound rule for modification of diet.

20. Saline /

20. Saline treatment, more especially with Sod. Sulphate, is an important factor in both Bacillary and Amoebic Dysentery. In the former it is specially important.
21. In Amoebic Dysentery the specific is Emetine in some form.
- Amelioration of symptoms is not to be confused with an actual cure, which means the eradication of the infecting parasite. A single course of 12 doses Emetine hypodermically may relieve the former, but for the latter it may be necessary to undergo prolonged treatment with Bismuth Emetine Iodide.
22. Even after most thorough treatment there is a small residue, composing permanent "carriers".
23. With efficient early treatment of Amoebic Dysentery with Emetine, Amoebic Abscess should not occur. When it does occur, further treatment may be necessary, such as aspirating the pus and injecting Emetine solution or a solution of Quin. Bihydrochlor. An open operation may be necessary.
24. In Bacillary Dysentery the value of early and efficient treatment with Anti-Dysenteric serum is recognised. Serum treatment is useless later in the course of the disease, unless for a relapse.

25. Anaphyllaxis may occur, but it can be guarded against by special method of administration in doubtful cases.
26. A sero vaccine has been employed prophylactically. Incidence of Dysentery was much less in inoculated than in uninoculated.
27. Vaccine treatment in combination with Serum treatment in the early stage of Bacillary Dysentery has met with success. No success is claimed for it in the chronic stage of the disease.
28. Dysenteric Arthritis develops in cases treated by serum originally, as well as in those who were not so treated; but where serum has been given early, the Arthritis is less likely to occur, and, if it does occur, appears to run a milder course.
29. Chief indications in treatment of Arthritis are aspiration of joint fluid and careful fixation of the joint. Later, massage and passive movements.

Apart from the diagnostic element in aspirating the fluid, aspiration should only be done if the joint is over-distended.

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APPENDIX.NOTES on CASES.Case 1.- Cpl. M.....

Dysentery B. Flexner
with Arthritis.

Admitted December 1917 suffering from a moderately acute attack of Dysentery B. Flexner. No serum was given, but Saline treatment adopted. Dysenteric condition progressed satisfactorily, but in the 4th week he developed an Arthritis - first, of the left knee associated with pain and synovitis. About 24 hours later the right knee commenced to swell also. The fluid being considerable in the left joint, it was aspirated. The fluid was sterile. Both joints were treated on lines indicated under treatment and made a good, though slow, recovery to normal. The patient became convalescent and was discharged well to Convalescent Camp after three months in Hospital.

In this case the Arthritis developed while still confined to bed.

Case 2. Private C.....

Dysentery, Clinical
with Arthritis.

Previous history of Rheumatic Fever.

History of Diarrhoea for 3 weeks previous to admission to Hospital with Clinical Dysentery in end of November 1917.

Subsequently he developed an Arthritis of the right knee, when he was transferred to - Hospital.

I first saw him in 3rd week of January 1918. There was then a chronic thickened condition of right knee with slight fluid. Movements of joint slightly restricted.

For two months he was under my care, during which time the bowels alternated between constipation and diarrhoea, but no blood in mucous. Stool examination negative. The joint condition improved slightly but, being of a chronic type, he was invalided and eventually discharged by Hospital ship.

No serum had been given in this case.

[illegible]

Previous History: Traumatic synovitis left knee.
No Rheumatic Fever.

Previous Dysentery: Moderately severe attack with relapse, September 1917.
No serum given; no joint trouble.

Shortly after leaving Convalescent Camp he was re-admitted to Hospital with an acute relapse, Novr. 1917. No serum given. Progress satisfactory and was allowed up 3 weeks later. Two days later Arthritis developed in left knee and he was put back to bed. Was transferred to Hospital and came under my care, January 1918. The joint then was practically free from swelling, but there was some stiffness and there was some pain in movement, especially on extension.

The bowel condition was unsatisfactory, as there was a tendency to occasional diarrhoea with slight mucous. Examination of stools were consistently negative though it was suggestive of being Amoebic.

He was invalided and discharged by Hospital ship eventually.

Case 4.

Private H.....

Dysentery B. Shiga.
Arthritis.

No history of previous Rheumatic Fever,
nor Dysentery.

Admitted to Hospital end of November 1917 with a moderately severe attack of Dysentery, proved to be due to B. Shiga. No serum given.

Three weeks later got up, but had to return to bed with Arthritis of the right knee. He felt pain in both hips and knee, but fluid only in right knee. He was transferred to ... Hospital and came under my care in the 3rd week of January 1918. The dysenteric bowel condition was cured, but the right knee was still swollen with fluid in the joint and tender to the touch. 5 cc. fluid was drawn off and proved to be quite sterile.

The joint improved slowly, but 2 months later there was still some fluid present.

He was invalided and discharged by Hospital ship.

Case 5.

Private J.....

Dysentery B. Shiga
with Arthritis.

Illness commenced early in June, 1918, with Diarrhoea, abdominal pain and irregular pyrexia. He was under observation in ... Hospital for Paratyphoid, but blood and mucous appearing in the stool was transferred to ... Hospital and came under my care 3 weeks from commencement of illness. He then had diarrhoea, several stools daily with only slight evidence of Dysentery. Slight pyrexia 98°-100° continued. Widal reaction indefinite - positive to low titre which previous T.A.B. inoculation would quite explain.

No serum was given at this point, owing to mild nature of Dysenteric symptoms and otherwise indefinite nature of the case.

Blood film = positive subtertian malaria. Intramuscular Quinine 20 gr. B.I.D. for a few days kept the temperature normal. Then about the 4th week from the appearance of blood and mucus in the stool the temperature again rose and Arthritis left knee developed with considerable effusion. The joint was aspirated and fluid was absolutely sterile. At this point Anti-dysenteric serum 40 cc. was given on three successive days.

The fluid reaccumulated in joint next day and the/

the right knee joint also became affected but was not tapped, as the distension was not great. Both joints were fixed in M'Intyre's splints and painted daily with Lin. Iod. In the course of a week the fluid subsided and the joints were normal. There was marked atrophy of muscles. The dysenteric condition did not improve. The stools were not more than 3 or 4 a day, but were distinctly dysenteric in character. B. Shiga was isolated. General toxæmia increased and, despite subcutaneous saline infusion, stimulation and Quinine treatment, he gradually got weaker, more emaciated, and died.

At the post-mortem: The whole bowel was in a most acute Dysenteric condition and quite out of proportion to the clinical evidence from the stool.

The knee joint was opened and appeared to be perfectly normal.

Case 6.

Private Mc.....

Dysentery B. Shiga
with Arthritis.

Admitted to Hospital, September 1918, with
a moderately severe Acute Dysentery.

Antidysenteric serum, 40 cc. for 3 successive days.

Serum reaction about 10th day with definitely painful
serum joints - no fluid. Slight pyrexia for a day
or so. This settled down quite and joints were quite
free of pain. About 3rd week complained of conjunc-
tivitis and slight purulent blepharitis. Both eyes
affected. A few days later Arthritis developed in
left knee. A moderate amount of fluid in joint.
Slight pyrexia rising to 100° and 101° for several
days when the temperature then settled down to 98-99°.

The joint was painful at first and treated with
lead and opium fomentations and fixed in splint.
Fluid aspirated -- sterile.

The joint condition improved and with massage
and gentle movements, became quite normal in the
course of a few weeks.

The Dysenteric condition - Stools semi-soft with
occasionally mucopus persisted.

Owing to the chronic and debilitated state of
the case generally, he was invalided and discharged
by Hospital ship in the end of October 1918.

The interesting feature of this case was the
association of Conjunctivitis preceding the Arthritis
and no gonorrhoeal history.

Case 7.

Gunner R.

Dysentery Clinical
with Arthritis.
(mixed infection.)

This man had severe compound comminuted fracture in the upper end right Tibia as a result of G.S.W. about September 22nd, 1917.

This was treated early and thoroughly. Anti-tetanic serum was given and repeated following week. Had severe Malarial relapse. Abscess developed popliteal space. This was opened and drained.

He then developed mild attack Clinical Dysentery after 2 weeks from being wounded and was transferred to Hospital and came under my care.

At this point the wounds were slightly purulent and there was an irregular pyrexia.

Serum was not given, as the dysenteric condition was mild and the condition of the pulse was poor. A few days later a metastatic superficial abscess developed over the left hip region. This was opened and drained.

A fortnight later Arthritis developed in the left knee - moderate amount of fluid and painful. At first treated with local applications and fixation. After 2 days the distension was greater and more painful, so the joint was aspirated and a purulent fluid withdrawn. This was full of streptococci.

The Dysenteric symptoms had cleared up and he was transferred for further surgical treatment to the original Hospital.

Further History: Joint did well; but gangrene developed later - first in one foot and then in the other. Eventually both limbs were amputated, but he was making a good recovery when last I heard.

Case 8.

Driver W.

Dysentery B. Shiga
with Arthritis.

Admitted June, 1918, with moderately acute attack of Clinical Dysentery. No serum given and no joint complication in first attack. During Convalescence had severe relapse, August 1918. Anti-dysenteric serum, 60 cc. for 3 days given. B. Shiga isolated. About 10th day had marked serum reaction including slight joint pains.

About the end of the 3rd week Conjunctivitis commenced - first in one eye, and then in the other, with slight iritis and marked photophobia.

Then joint symptoms developed, first in left knee, and then in right, associated with irregular pyrexia 98°-100° for several days. The temperature had previously been keeping normal. There was very little fluid swelling of the joints, but pain was more pronounced.

Both the conjunctivitis and Arthritis gradually subsided, and he became convalescent again in the course of two weeks. On getting up there was a very marked oedema and anasarca of the lower limbs. Urine was normal. He was invalided by Hospital ship.

The joint was not aspirated in this case, as there was very slight effusion.

The interesting feature of this case was the Conjunctivitis preceding the Arthritis.

Case 9.

Driver S.

Dysentery Clinical
with Arthritis.

After having only been one week in Salonica he was admitted to Hospital with an acute attack of Dysentery in August 1918. Neither Bacilli nor Entamoebae were discovered in the stool. Anti-dysenteric serum 40 cc. for 3 successive days was given. Later, in convalescence, about the 4th week, while still in bed, he developed an Arthritic left knee with considerable fluid effusion and slight pyrexia, 98°-100°. The joint was aspirated and fluid was sterile. The pyrexia lasted for several days, but the joint gradually recovered and became quite normal again.

Shortly after this, before he had got up, he had another severe relapse of Dysentery. Serum was again given. As a very marked serum rash appeared shortly after it was given, it was not repeated. The Dysenteric condition rapidly improved. There was no recrudescence of the Arthritis with the second attack of Dysentery.

Though originally of good physique he became anaemic and debilitated, and was invalided by Hospital ship, while convalescent from his second attack.

Both attacks were very well marked clinically, but no positive finding could be obtained from the stools.

Case 10.

Private D. Dys. B. Shiga with
Cerebral Haemorrhage.

Previous History: Good before going East.
Dysentery, August 1917.
Malaria: 1st attack, July 1917.
Relapses, August and Novr. 1917.

History. Admitted ... Hospital, Nov. 29th 1917, with
Malaria Clinical. Diarrhoea commenced, Dec. 19th;
20 stools in 24 hours,- the last contained blood and
mucus. Transferred to Hospital December 19th
with Acute Dysentery. Stools very frequent - pure
blood and mucus. Temperature 101°.

On examination: Heart normal; Lungs normal; Spleen
palpable and tender; Abdomen, tender over lower part.
Blood film - negative.

Progress: Anti-dysenteric Serum 40 cc. given sub-
cutaneously on evening of admission and two following
days. Usual Sod. Sulph. treatment by mouth.

Intramuscular Quinine 20 gr. twice daily during acute
dysenteric period. B. Shiga isolated from the stool.

26-12-17: Progress good; stool now liquid faecal
with mucus.

29-12-17: Stool, blood-stained mucopus.

12- 1-18: Sloughs coming away in stool.

23- 1-18: First showed evidence of being drowsy,
groaning and restless.
Incontinence of urine. Eye reflexes
normal, some mystagmus, no paresis.

25- 1-18: Semicomatose. Pupils equal, dilated
and sluggish to light. Temperature
101.4°. Pulse 120.
Stool soft, without blood or mucus.

26- 1-18: Unconscious. Pupils react; Knee reflexes absent; Plantar (flexor) present. Urine Cath: Spec:- no sugar, some mucus, trace albumen. No acetone nor Diacetic acid.

Intravenous: Saline with Quin. Bihydrochlor. gr.20 given and repeated same evening and repeated daily till 29-1-18.

Never regained consciousness and died 31-1-18.

There was never any evidence of convulsion.

Latterly stool improved and was occasionally semi-soft and free from blood and mucus.

In this case the man was prematurely old.

Though only 28 he had marked arteriosclerosis.

A Wasserman reaction was taken and proved negative.

When the cerebral symptoms developed it suggested Cerebral Malaria in the light of the malarial history and splenic enlargement, though blood films were negative. Intravenous Quinine was then started.

Post Mortem: There were advanced dysenteric lesions in the bowel, but the unique condition was a haemorrhage into the anterior part of the caudate nucleus on the left side. There was no evidence of malarial parasites microscopically in the brain.

Case 11.

Private B.

Amoebic Dysentery with
Intestinal Haemorrhage.

Had been under observation at periodic intervals, for as long as six weeks at a time, with Chronic Diarrhoea. Frequent examinations had been made, but the stool was never found positive.

Was in -- Hospital, January 1918, with recurrence of Diarrhoea. There was also some thickening and tenderness in the region of the appendix, suggesting Chronic Appendicitis.

Appendicostomy was performed successfully. The following day there was a severe intestinal haemorrhage - three bedpans full of dark blood clot were passed at intervals. The first specimen passed was sent to the laboratory for examination, and Entamoeba Histolytica was discovered to be present. General treatment adopted - foot of bed raised; Calc. Chloride 10 gr. four hourly by mouth, and Morph. $\frac{1}{4}$ gr. hypodermically. Pituitrin 1 cc. Adrenalin Sol. 1-1000 by bowel. Emetine hypodermically 1 gr. commenced same evening. Next day, mere trace of blood clot, and no further haemorrhage later. Kept on regular Emetine treatment $1\frac{1}{2}$ gr. daily, and made an excellent recovery.

This case is interesting, inasmuch as the infection was only proved on the occurrence of the haemorrhage.